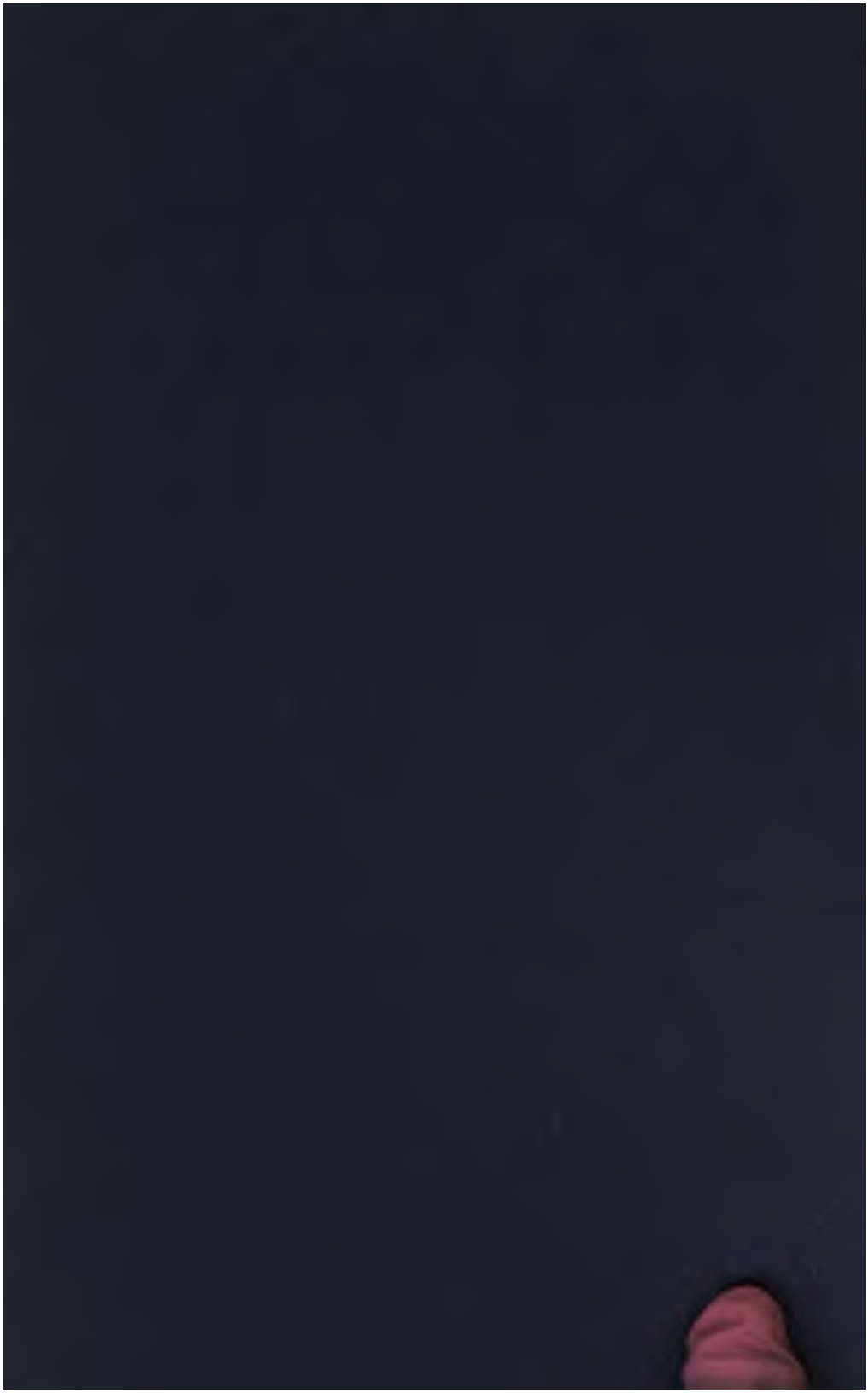
NERVOUS DISEASES

STARR



SYNOPSIS OF LECTURES

UPON

Diseases of the Nervous System,

DELIVERED AT THE

COLLEGE OF PHYSICIANS AND SURGEONS,
MEDICAL DEPARTMENT OF COLUMBIA UNIVERSITY,
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SYNOPSIS OF LECTURES

UPON

DISEASES OF THE NERVOUS SYSTEM.

A KNOWLEDGE of the structure and functions of the central nervous system is an essential preliminary to the study of its diseases. The human nervous system is the most complex of all nervous structures, and is the culmination of the process of evolution. It will aid in the understanding of its structure if the various stages in its evolution are known.

THE EVOLUTION OF THE NERVOUS SYSTEM.

The simplest form of a nervous system consists of a single mass of gray matter with a sensory and a motor nerve.

The mass of gray matter receives impulses sent from the surface by the sensory fibre, acts in response to the impulse, and the action is transmitted to a motor mechanism by the motor nerve.

Example. The closure of an oyster-shell when the oyster feels any object touching its sensory surface. Parallel action in human system—any spinal reflex—*e.g.*, knee-jerk.

Several such single masses may be joined to one another, each with its sensory and motor nerve. The masses receive impulses in succession and act in succession.

Example. Wave-like motion of a jelly-fish or caterpillar.

A compound type of nervous system consists of several subordinate masses joined together and presided over by a single higher mass, which is joined to each of the lower masses. The higher mass receives impulses from the lower masses, not directly from the surface. It sends impulses to the motor mechanisms through the lower masses, not directly. It secures a certain definite succession and combination of action in the lower masses.

Examples. The nervous system of a frog experimentally deprived of its hemispheres, capable of automatic acts of swimming. The automatic act of respiration in man. In man the facial, laryngeal, intercostal, thoracic, and diaphragmatic respiratory muscles act in definite succession and rhythm in the act of breathing; and this act is presided over by a single centre in the medulla controlling the lower medullary and spinal centres.

The highest type of nervous system is the complex type. There are the lower centres as in the simple type. There are higher centres controlling the lower centres, as in the compound type. There is a supreme mass controlling both the others.

This is the form found in all vertebrates, and the degree of evolution in the highest or supreme mass determines the place of the animal in the scale of intelligence.

In man the supreme mass is the cerebral cortex, the next lower or automatic centres are the optic thalami and corpora striata, including gray masses in the cerebral axis; the lowest or reflex centres are the cranial nerve nuclei in the cerebral axis and the gray matter of the spinal-cord segments.

These distinct gray masses are joined with one another in all possible combinations by means of the white nerve tracts, which pass in all directions around and within them.

Read Herbert Spencer, ' Principles of Psychology," Part I., Chaps. I.–IV.
Charles Mercier, "The Nervous System and the Mind," Part I.

The Neurone.

The primary elements of the nervous system are neurones, and the nervous system from the simplest to the most complex in the order of evolution consists of a collection of such neurones of greater or lesser complexity.

The neurone consists of:

(1st) A cell body whose shape and size vary at different parts of the nervous system.

(2d) Dendrites or protoplasmic prolongations of the cell body which divide and subdivide as they extend outward from it much like the roots of a tree. The surface of these dendrites is rough, appearing like moss under the microscope on account of the

presence of fine granules. Nervous impluses are collected by these dendrites and transmitted to the cell body. Fine filaments have been demonstrated passing from each dendrite through the cell body and entering the axon.

(3d) The axon or axis-cylinder process of the cell body is a single long, projecting fibre passing outward from the cell body and transmitting impulses from the cell body to a distance. The neuraxon gives off fine collateral branches in its course and terminates as do its collaterals in a fine brush-like end. This brush is supposed to come in contact with the gemmules of the dendrites of other neurones so that connection between different neurones is possible. The axon is made up of many fine fibrillæ which, as already stated, have come through the cell from the dendrites. The cell body controls nutrition of the dendrites and axon so that when it is destroyed they atrophy. Golgi divides neurones into two types. In the first type the axon extends to a distance and ends in a single brush. In the second type the axon divide into numerous branches near the cell body and ends in a network of fibres.

The Structure of a Neurone.—A fine trabeculum or framework appears to be filled with semifluid protoplasm, which protoplasm is used up by action of the neurone, thus leading to alterations in size in accordance with the amount of work performed. The normal cell body shows under straining with methyl blue a granular appearance, and these granules are termed chlorophyl bodies and appear to have a regular arrangement within the cell. The protoplasm within the trabeculæ does not stain. In a degenerated cell marked changes in the staining capacity and an irregularity of arrangement of the chlorophyl bodies are noticed.

Varieties of Neurones.—Neurones have been divided according to their function into : (1st) Centrifugal, which are either (*a*) *motor*, (*b*) *secreting*, (*c*) *trophic*. (2d) Centripetal, or sensory. (3d) Intrinsic, or association. Neurones have also been divided into a series according to their position, into (1) primary neurones whose cell body lies in the central nervous system or in the ganglia, and whose axon extends thence to some peripheral portion of the body, *e.g.*, to a muscle, to a gland, to

the skin; (2) secondary neurones whose body lies in the central nervous organs and whose axon extends thence to some other part of the central nervous organs to terminate about a primary neurone. These neurones may lie in the cortex and brain or in the basal ganglia or medulla.

All simple reflex acts involve the transmission of impulses through primary neurones only.

All transmission of voluntary motor impulses, or conscious sensory impulses, involves the action of both primary and secondary neurones.

The human nervous system has two distinct portions: (1) the cerebro-spinal system, and (2) the sympathetic nervous system.

(1) The cerebro-spinal system includes: (*a*) a peripheral portion, comprising the nerves, (*b*) a central system, comprising: (i) the spinal cord and cranial nerve nuceli; (ii) the cerebellum gray masses of the medulla, pons, crura cerebri, corpora quadrigemina, optic thalami and corpora striata; (iii) the cerebral cortex.

(2) The sympathetic nervous system is subdivided into: (*a*) Two great cords containing ganglia, which lie on the sides of the vertebral column and are joined to the spinal cord on one side and to the plexuses on the other; (*b*) Three pervertebral plexuses, the cardiac, solar, and hypogastric, which are masses of ganglia connected with the viscera; (*c*) Many peripheral plexuses connected with various organs; (*d*) Terminal monocellular ganglia scattered through the viscera; (*e*) Sympathetic nerve-fibres joining the plexuses and ganglia together and connecting them with the cerebro-spinal system.

THE PERIPHERAL NERVES AND THEIR DISEASES.

STRUCTURE of the nerves. Bundles of nerve-fibres—large or small.

Structure of a nerve-fibre: 1. Axis cylinder, a long process of some nerve-cell. 2. Medullary sheath of myelin—a fatty insulator, interrupted at the nodes of Ranvier. 3. Connective-tissue sheath of Schwann, with its nucleus. Nerve-fibres are bound into bundles by connective tissue; endoneurium, perineurium. The sympathetic nerve-fibres have no medullary sheath.

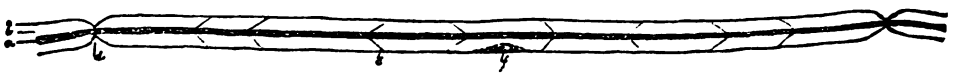


FIG. 1.—A Normal Nerve,

All nerve-fibres branch during their course in the central nervous system, sending out "collaterals," but do not branch in their peripheral course. They arise from a nerve-cell. They terminate in fine brush-like expansions which are spread out in the skin, or on a muscle, or around another nerve-cell.

Nerves are divided into motor nerves and sensory nerves, according to the mechanism to which they are attached. Motor nerves convey impulses outward, sensory inward. Motor nerves grow outward from large nerve-cells in the spinal cord. Sensory nerves grow from cells in the posterior spinal ganglia, which send one branch to the surface, and the other into the spinal cord.

NEURITIS.

Neuritis may occur in a single nerve, or in a plexus.

Causes.—Injuries, strains, pressure, compression in fractures, rheumatism, gout, cold.

Pathology.—1. Parenchymatous neuritis, congestion and exu-

dation in nerve-trunk; swelling and degeneration of individual fibres; (1) axis cylinder broken and disintegrated in fatty mass; (2) medullary substance separated into segments and fatty globules, and mingled with granules and nucleated cells; (3) connective-tissue sheath shrunken and empty. Nuclei multiply. Products of degeneration liquefy and are absorbed.

FIG. 2.—Neuritis.

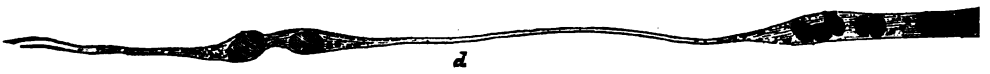


FIG. 3.—Neuritis.

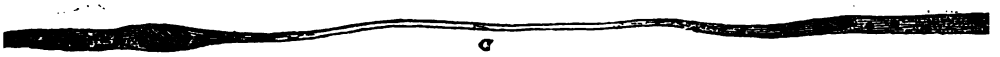


FIG. 4.—Neuritis.

Regeneration occurs by growth of new fibres outward from the healthy stump into the old sheath.

2. Interstitial neuritis. The connective tissue of the endoneurium and perineurium increases in volume and the nerve-fibres are compressed by the congested vessels and by the new tissue. These forms usually occur together in non-traumatic cases.

Symptoms.—Weakness or paralysis with atrophy and reaction of degeneration in muscles supplied by nerves involved. Reaction of degeneration (R. D.) is a change in the action of the nerves and muscles to the electric currents. The response of the nerve to both currents is lost. The muscle loses its faradic reaction. The galvanic reaction is changed, the muscle responding more actively to the closure of the positive pole (anode) than to that of the negative pole (kathode). Pain numbness or anæsthesia in skin supplied by nerve involved. Vasomotor and trophic changes in the distribution of nerve involved. Coldness from

sluggish metabolism; glossy skin; œdema; pain and tenderness at seat of inflammation in interstitial cases, and along the nerve trunk.

Course.—Slow spontaneous recovery when continuity of nerve is preserved. Otherwise, no recovery.

Treatment.—If nerve is broken unite divided ends. Rest the part injured but exercise the muscles paralyzed by electricity and massage. Use hot applications, sedative lotions. Protect from cold by cotton batting. Morphine for pain.

Multiple Neuritis.

Multiple neuritis is an inflammatory or degenerative disease of the peripheral nervous system varying in extent and intensity and affecting symmetrical parts of the body.

The motor nerves may be involved alone (*e.g.*, in lead and mercury poisoning), or the sensory nerves alone (*e.g.*, in coal-gas poisoning), or both may be involved together (*e.g.*, in alcoholic neuritis). The distal parts of the nerves are commonly affected to a greater extent than the central parts. Hence the symptoms appear chiefly in the distal parts of the extremities.

The pathological process is the same as in simple neuritis and may be parenchymatous alone, or interstitial alone, or both.

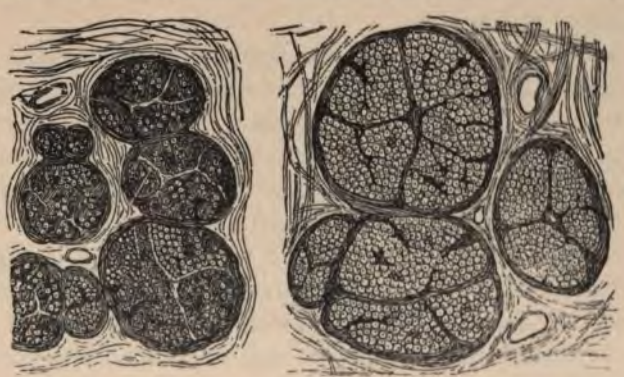


Fig. 5—Neuritis contracted with normal nerve.

Etiology.—1. Toxic cases: Alcohol, lead, arsenic, bisulphide of carbon, copper, mercury, phosphorus, coal gas, coal tar drugs.

2. Infectious cases: Diphtheria, typhoid, typhus, scarlet fever, measles, malaria, leprosy, small-pox, erysipelas, gonorrhea, grippe, and beri-beri.

3. Rheumatic cases: Exposure to cold, overexertion.

4. Secondary to anæmia, gout, tuberculosis, syphilis, septicæmia, general malnutrition, and diabetes.

Symptoms.—Acute or subacute onset with fever, 103°-104° F., and general febrile symptoms—sometimes without fever.

Sensory Symptoms.—First to occur. Pain—sharp, severe, located in limbs, increased by motion or pressure; muscular and nerve tenderness, sensitiveness.

Paræsthesiæ—Burning, tingling, numbness, band about legs and body.

Anæsthesia—In tips of fingers and toes, extends up limbs.

Ataxia—In fine movements and loss of sense of position.

Motor Symptoms.—Paralysis, in extensors, dropped wrist and foot, general inability to walk or move in bed; contractures; cranial nerves may be paralyzed, causing symptoms in face and eyes.

Atrophy—Rapid in onset. R. D., or diminution in electric contractility; loss of deep reflexes; no paralysis of sphincters.

Trophic Symptoms.—Vasomotor paralysis; glossy skin; œdema; abnormal growth of nails and hair; sweating profuse; no bedsores; urticaria.

Certain cases present peculiar features in their course.

1. *Alcoholic Neuritis.*—(*a*) Paralytic type. Rapid onset is the rule, with delirium and delusions, subsequently imperfect memory of recent events. Severe paralysis in extremities with very great pain; anæsthesia very marked in hands and feet, also tremor and tenderness; dropped wrists and feet; or (*b*) Ataxic type. Sharp pains are soon followed by great ataxia in legs and hands, by anæsthesia and partial paralysis; but no loss of control of bladder or rectum. In both forms there is danger of heart failure or death from exhaustion, due to complicating gastritis. Usually there is rapid increase of symptoms for three or four weeks, and then, after a stationary period of two months, a slow progress to recovery, the duration being from six to twelve months.

2. *Lead Neuritis.*—Onset with intestinal colic, lead line on gums. Paralysis causing dropped wrists, without sensory disturbance in hands, Deltoids next paralyzed. May affect all muscles of arms and legs, and go on to sensory symptoms with delusions, but this is rare.

Gradual recovery, when poison is eliminated, in four months.

3. *Arsenical Cases.*—Gastric disturbance at outset. Then paralysis with ataxia, tremor, atrophy, and great numbness, but little pain; legs and arms equally affected. Skin darkened. Recovery in two to six months, depending on original severity. Contractures.

4. *Coal-Gas Cases.*—Subacute and slight, chiefly sensory, numbness for a long time in fingers, hands, and feet.

5. *Diphtheritic Cases.*—Severe paralysis of all extremities, usually without sensory disturbance, cranial nerves frequently affected. It begins in uvula, and swallowing and talking become difficult. Eyes are often turned. Ataxia is marked in some cases. Never pain. Progress to recovery usual in three months.

6. *Epidemic Form.* (Beri-beri.) Occurs in Brazil, India, Borneo, Japan, Philippines. Several types: acute pernicious, chronic œdematous, chronic atrophic. Too rare to need attention here. (See Organic Nervous Diseases. Starr. Chap. VIII.)

Diagnosis of Multiple Neuritis.—From anterior poliomyelitis: by pain, tenderness along nerves, sensory symptoms, distribution of paralysis, symmetrically. From locomotor ataxia: by rapid onset and paralysis, preservation of control of bladder, and absence of Argyle-Robertson pupil. From myelitis: by absence of affection of bladder and rectum, by absence of bedsores, and lack of tenderness of spine to heat, and by distribution of symptoms in periphery.

Prognosis.—Good in large majority of cases; slow recovery; bad when heart becomes rapid and respiration poor.

Treatment.—Rest in bed. Warm applications to limbs by packs. Rubbing with oil; gentle massage if possible. Prevent deformities and contractures by proper position of limbs. Electricity, galvanism through nerves and to muscles after acute stage. Baths, warm, 98° F., for half-hour several times daily.

Attention to diet, nourish well, fatty food, cut off alcohol in all cases.

General and nerve tonics; iron, quinine, strychnine, glycerophosphate of lime and soda

In acute stage, sodium salicylate, gr. x., q. 3 hr., and sedatives, morphine, phenacetine.

In chronic stage, arsenic $\frac{1}{60}$ gr. t.i.d., cod-liver oil.

NEURALGIA.

Neuralgia is a painful affection of the sensory neurone, sometimes functional, sometimes due to organic changes in the sensory ganglia.

Causes.—Same as neuritis.

Symptoms.—Spontaneous pain of intense character in the course and termination of a single nerve. The pain occurs in paroxysms, is not continuous like neuritis. Painful points, tender to pressure may be found, but the entire nerve is not tender as in neuritis. Herpes Zoster often accompanies neuralgia.

Varieties as numerous as the nerves. Trigeminal, Brachial Intercostal Sciatic most common.

Prognosis.—Good except in organic cases and in trigeminal and sciatic, where recurrence is the rule.

Treatment.—Remove cause, if found. Locally heat, counter-irritation, salicylates, coal-tar drugs, opium, gelsemium, division of nerves, exsection of ganglia.

THE SPINAL CORD AND CRANIAL-NERVE NUCLEI.

THE spinal cord in man is made up of thirty-one segments—each of which consists of a mass of gray matter joined to a pair of spinal nerves. There is no apparent line of boundary between adjacent segments, the cord being a long cylindrical organ. But in snakes and rabbits the area of gray matter is much larger opposite the entrance of nerves than between them, and in fishes there is a visible boundary between adjacent segments.

Each segment presents a symmetrical arrangement of its two halves. Each half consists of a central mass of gray matter surrounded by white tracts. Each gray mass has an anterior horn, a central mass, and a posterior horn. To each horn nerve-fibres are joined, making the anterior and posterior nerve-roots. The two halves are separated from one another in front by the anterior fissure, and behind by the posterior septum. They are joined together by the anterior white commissure and by the gray commissure in which lies the central canal of the cord. The functions of each segment are shown in the table on page 14.

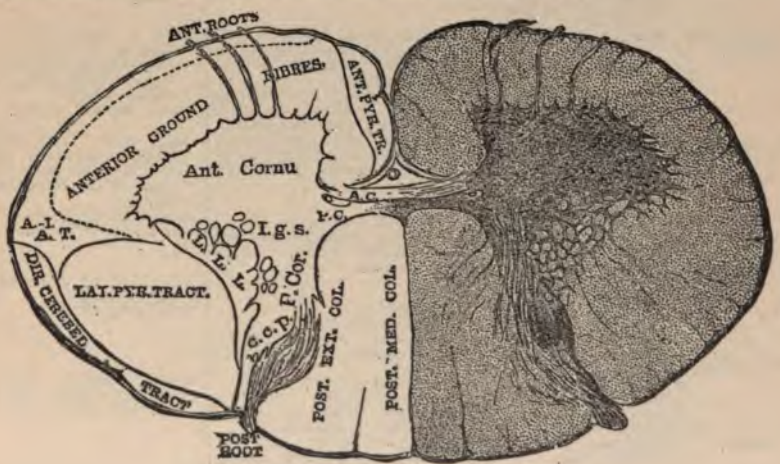


FIG. 6.—Cross Section of Spinal Cord. (Gowers)

Segment.	Muscles.	Reflex.	Sensation.
2d and 3d Cervical.	Sterno-mastoid. Trapezius. Scaleni and neck. Diaphragm.	Hypochondrium. Sudden inspiration produced by sudden pressure beneath the lower border of ribs.	Back of head to vertex. Neck.
4th Cervical.	Diaphragm. Deltoid. Biceps. Coraco - brachialis. Supinator Longus. Rhomboid. Supra- & infra-spinatus.	Pupil 4-7 cervical. Dilatation of the pupil produced by irritation of neck.	Neck. Upper shoulder. Outer arm.
5th Cervical.	Deltoid. Biceps Coraco-brachialis Brachialis anticus. Supinator longus. Supinator brevis. Deep muscles of shoulder blade. Rhomboid. Teres minor. Pectoralis (clav. part). Serratus magnus.	Scapular. 5th Cervical to 1st Dorsal. Irritation of skin over the scapula produces contraction of the scapular muscles. Supinator Longus. Tapping its tendon in wrist produces flexion of forearm.	Back of shoulder and arm. Outer side of arm and forearm. Anterior upper two-thirds of arm.
6th Cervical.	Brach. anticus. Pectoralis (clav. part). Serratus magnus. Triceps. Extensors of wrist and fingers. Pronators.	Triceps. 5th to 6th Cervical. Tapping elbow tendon produces extension of forearm. Posterior wrist. 6th to 8th Cervical. Tapping tendons causes extension of hand.	Outer side of arm and forearm. Inside and front of forearm.
7th Cervical.	Triceps (long head). Extensors of wrist and fingers. Pronators of wrist. Flexors of wrist. Subscapular. Pectoralis (costal part). Latissimus dorsi. Teres major.	Anterior wrist. 7th to 8th Cervical. Tapping anterior tendon causes flexion of wrist. Palmar. 7th Cerv.-1st Dor. Stroking palm causes closure of fingers.	Inner and back of arm and forearm. Radial distribution in the hand.
8th Cervical.	Flexors of wrist and fingers. Intrinsic muscles of hand.		Forearm and hand; median and ulnar areas.
1st Dorsal.	Extensors of thumb. Intrinsic hand muscles. Thenar and hypothenar eminences.		Ulnar distribution to hand.
2d and 12th Dorsal.	Muscles of back and abdomen. Erectores spinae.	Epigastric. 4th-7th Dorsal. Tickling mammary region causes retraction of epigastrium. Abdominal. 7th-11th Dorsal Stroking side of abdomen causes retraction of belly.	Skin of chest and abdomen, in bands running around and downward corresponding to spinal nerves. Upper gluteal region.
1st Lumbar.	Ilio-psoas. Sartorius. Quadratus lumb. Transversalis.	Cremasteric. 1st-3d Lumbar Stroking inner thigh causes retraction of scrotum.	Skin over groin and front of scrotum, and narrow band down the front of the thigh and leg.
2d Lumbar.	Ilio-psoas. Sartorius. Flexors of knee (Remak) Quadriceps femoris.	Patella tendon. Striking tendon causes extension of leg. 2d-4th Lumbar.	Inner side of thigh. Outer side of thigh.
3d Lumbar.	Quadriceps femoris. Inner rotators of thigh. Adductors of thigh.		Inner side of thigh and leg to ankle.
4th Lumbar.	Abductors of thigh. Adductors of thigh. Flexors of knee (Ferrier) Tibialis anticus.	Gluteal. 4th-5th Lumbar. Stroking buttock causes dimpling in fold of buttock.	Inner side of foot.
5th Lumbar.	Glutei. Biceps femoris. Semi-tendinosis. Outward rotators.	Achilles tendon. Over-extension causes rapid flexion of ankle, called ankle clonus.	Lower gluteal region back of thigh. Leg and foot outer part.
1st and 2d Sacral.	Flexors of ankle. Long flexor of toes. Tibialis post.	Babinski reflex. Scratching sole of foot causes retraction of great toe. Plantar. Tickling sole of foot causes flexion of toes and retraction of leg.	Leg and foot except inner side.
3d Sacral.	Peroneus longus. Intrinsic muscles of foot		
4th and 5th Sacral.	*Sphincter ani et vesicae.*	Bladder and rectal centre.	Perineum and back of scrotum. Anus.

The Anterior Horns of the Spinal Cord.

These vary greatly in size and shape in various segments, being large opposite the entrance of the cervical and lumbo-sacral nerves and thus causing the cervical and lumbo-sacral enlargements of the cord.

Their size and shape depend upon the number of groups of cells found at different levels; for the horns are made up of cells —large, polygonal cells with nucleus and nucleolus—collected into distinct groups.

Some groups of cells lie on the edge of the horn, making projections of the gray mass into the white columns. These groups are found in all the higher vertebrates and are not peculiar to man. Other groups lie within the horn and these are peculiar to man and the higher apes. Each group of cells presides over the action of a muscle and maintains its tone and nutrition, so that all motor impulses to the muscle, whether reflex, automatic, or voluntary, proceed from the group of cells. The outer groups govern muscles of flexion, extension, pronation, and supination of the larger joints. The inner groups govern the small muscles of the fingers and toes. Hence the outer groups are said to preside over fundamental motions and the inner groups over accessory motions.

The thumbless monkey has no inner groups in the cervical region. These are present in monkeys with thumbs and in man. When the inner groups are destroyed by disease, the finger motions are paralyzed.

In each segment there may be from two to eight groups of cells. Any group may extend through several segments. If a single segment is destroyed by disease several muscles will be paralyzed. But no muscle will be totally paralyzed unless its entire group of cells is destroyed. Total paralysis with complete atrophy in a muscle indicates destruction of the entire group of cells which govern the muscle. If the group extends through several segments the paralysis implies disease in all the segments. The table shows the situation in the various segments of the group of cells governing the various muscles, so far as these have been determined.

Anterior Poliomyelitis.

Anterior Poliomyelitis is a disease limited to the anterior horns of the spinal cord. It may be acute or chronic.

Acute anterior poliomyelitis is a common disease of childhood known as *infantile paralysis.* It occasionally occurs in adults. Chronic anterior poliomyelitis is an uncommon disease of adult life or old age, known as atrophic paralysis.

Acute Anterior Poliomyelitis. Etiology.—Occurs chiefly between 2 and 5 years, when exertion is greatest, in the summer, June to September, when exposure is greatest to heat, occasionally after falls and after fevers; it is probably an acute infectious disease in two-thirds of the cases. In the other third it is probably due to hemorrhage from or to thrombosis in a spinal blood-vessel.

Pathology.—Lesion is an acute inflammation of the entire gray matter of the anterior horn, the cells and interlacing fibres being affected. The cells become swollen, undergo degeneration, and liquefy, and then shrivel and atrophy. The fibres are separated from their cells and atrophy. The neuroglia is infiltrated with inflammatory corpuscles. The capillaries are distended and frequently rupture. The process may go on to complete destruction of the cells or may be arrested at any stage.

If it is arrested early, repair may ensue and the cells regain their former appearance. If it goes on, the result is a diminution in the size of the horn, a change in its shape, and a contraction of the entire cord at the level of the lesion.

As a rule the entire anterior horn is at first involved, but the permanent destruction is limited to one or two groups of cells on one side only.

Inasmuch as each motor nerve-fibre comes from and is a part of the motor cell, the nerve-fibres degenerate when the motor-cells are destroyed. Atrophy of the motor nerve-roots and motor-nerves from the anterior horns to the muscles is therefore part of the lesion in anterior poliomyelitis. The deformity of the spinal cord at the level of the lesion and the atrophy of the nerve-trunks are due in part to these changes in the nerve-fibres.

The muscles which are paralyzed atrophy rapidly, and when recovery does not occur they remain small.

While no special groups of cells are known to control the nutrition of the bones, it is found that if the gray matter near the central region of the segment is destroyed by the disease, the bones are much retarded in their growth. Hence in a severe case of infantile palsy the limb affected is smaller than other limbs in all its parts. But trophic changes in the skin, such as sores, glossy skin, or gangrene, do not occur. Through the anterior horns pass fibres from the central region which control the vascular tone and metabolic processes. Hence in lesions of the anterior horn symptoms due to the destruction of these fibres ensue, viz., sluggish circulation, cyanosis, and lowered temperature, with imperfect nutrition.

Symptoms.—Sudden onset with fever 100° to 103° F., occasionally without. Febrile symptoms: Headache, loss of appetite, nausea, vomiting, diarrhœa. Restlessness: often convulsions and delirium. The fever rarely continues more than two to three days. Pain in the back and limbs is complained of when child is old enough to talk.

Paralysis appears suddenly. Sometimes is found in the morning; is complete and extensive either in one limb or more; legs oftener than arms, 4 to 1; one limb more than the other. Maximum is reached in first week. Remains stationary for two to six weeks and then improves. Result is recovery, which is partial; some muscles remain paralyzed. The affected limb atrophies rapidly. Atrophy is most intense in the muscle permanently weak. There is a change in the electric reactions in the paralyzed muscles. R. D. Loss of faradic excitement in two weeks or sooner. Increase at first, then decrease, in galvanic excitability. Muscles contract slowly instead of quickly, more forcibly to the positive than to the negative pole.

The nutrition of the entire limb suffers; bones small and growth impaired; fat is less firm, and skin is cold; but no bedsores appear. The sphincters are not affected. The reflex action is lost at the level affected, not changed elsewhere.

Course.—There is a progressive improvement which begins

within two months, but usually some muscles remain paralyzed and atrophied, and the joints moved by these may become deformed; spine curved. The muscles most frequently affected are: The peronei and tibialis anticus in the leg—hence talipes equinus and varus; the muscles of the calf and anterior part of the thigh; the deltoid, biceps, supinators, and spinati in the arm—hence subluxation of humerus; the muscles of the hand and forearm.

General health is unaffected.

In adults acute anterior poliomyelitis presents the same symptoms, but fever is less severe and pain is greater in muscles. Extent of initial paralysis is greater. Occasionally the onset is slow, so that case is called subacute, developing in two to four weeks progressively, remaining for a time stationary and then subsiding.

Diagnosis.—From acute rheumatism in infants. Rheumatism may produce immobility of joints. It also causes pain, redness, swelling of joints, and tenderness.

Prognosis.—Good as to life.

As to recovery, at *end of two weeks* test faradic reaction. The muscles in which it persists will recover entirely.

At *end of three months* test again. The muscles in which it has returned will recover partially.

At *end of six months* test again. The muscles which do not respond will never recover entirely, but if galvanic reaction is good may improve.

As to use of limb, it depends on growth of bone; *e. g.*, short leg; on development of deformity and contractures; *e.g.*, talipes.

Treatment.—In acute stage: Cup or blister the spine and keep quiet. Give ergot and bromide, and antipyretics.

In chronic stage: Rubbing, warm clothing, galvanism to the muscles, interrupted, continuous. Strychnine $\frac{1}{100}$ to $\frac{1}{60}$ t.i.d., according to age. Prevent or remedy deformities by apparatus or surgical operation.

Chronic Anterior Poliomyelitis.—Progressive muscular atrophy occurs in adults between thirty and fifty years; is a rare disease.

Etiology.—Falls and injuries, exposure to damp and cold, alcoholic and sexual excesses, lead poisoning (?).

Symptoms.—Gradually increasing atrophic paralysis, several months in onset, preceded by fatigue and weakness and loss of weight. Muscles become flabby, then atrophied and paralyzed.

It begins in legs and extends upward (peroneal type).

It may begin in hands and then extend to shoulders and invade the back (Aran-Duchenne type).

Finally, all the muscles of the body, including face and tongue, may be affected. The tendon reflexes are lost.

There are fibrillary twitchings in muscles.

There are electrical changes which vary in different cases; usually R. D. is found.

The circulation is poor in the limb, skin cold and blue.

There is no sensory disturbance except slight numbness.

There are no visceral symptoms, no disturbance of bladder and rectal control. Bedsores do not develop.

Course.—Gradual increase after a long period of stationary state. Sometimes a steady progress to bulbar paralysis and death.

Treatment.—Similar to chronic stage of acute anterior poliomyelitis.

Cranial-Nerve Nuclei.

The groups of cells in the cerebral axis (medulla oblongata, pons Varolii, and crura cerebri) which give origin to the motor cranial nerves lie party on the floor of the fourth ventricle (XII.,

Fig. 7.—Longitudinal Section Showing the Relative Position of the Cranial Nerve Nuclei (Edinger).

VI., IV., III.), and partly deep beneath the floor in the formatio reticularis (XI., IX., VII., Vm.). These groups of cells are quite homologous to the groups in the anterior horns of the cord, and are subject to diseases whose pathology is the same of that of poliomyelitis.

Bulbar Paralysis—or Glosso-Labio-Laryngeal Paralysis. (*Rare*).

Symptoms.—Slowly progressing paralysis with atrophy in the muscles supplied by the motor cranial nerve nuclei.

1. Disturbance of speech. Alalia, tongue letters, R L D T, due to paralysis of XII. nerve nucleus. Atrophy and tremor of tongue with paralysis. Chewing, swallowing impaired.

2. Muscles of lips and face become weak and thin, due to paralysis of VII. nerve nucleus. Alalia extends to lip letters, P B V F. Atrophy and tremor of lips with paralysis. Whistling, facial expression impaired. Saliva runs and is increased.

3. Muscles of pharynx and larynx affected, due to paralysis of the IX. and XI. nerve nuclei. Food regurgitates or cannot be swallowed; reflex action impaired; larynx unprotected; voice monotonous, low; cough impossible.

There is no change of sensation or of taste.

Pulse sometimes rapid, 100 to 130.

Duration, two to five years.

Death from inanition, pneumonia, heart failure.

Ophthalmoplegia.

This is a rare disease, due to an atrophy of the nuclei of the nerves moving the eyeball or to hemorrhage into them.

The muscular apparatus of the eye consists of external muscles moving the eyeball: Recti and obliqui and levator palpebræ. Internal muscles moving the iris: Dilators and constrictors. Each of these may be affected alone or together.

Ophthalmoplegia { externa. / interna.

Ophthalmoplegia Externa. Symptoms.—Gradually increas-

ing loss of power in all the muscles of eyeball. One or more muscles at first, then others, till all are affected.

Result, ptosis. Various forms of strabismus, nystagmus, and finally immobility of eyeball. Double vision.

Ophthalmoplegia Interna. Symptoms.—Loss of reflex to light. Occurs as a symptom in locomotor ataxia and general paresis.

Diagnosis.—From paralysis of single nerves, III. nerve or VI. nerve, by the fact that such disease is unilateral and all the muscles supplied by the single nerve are involved, others remaining unaffected, and by the fact that the pupil is always affected.

Progressive Muscular Dystrophy.

This is a primary muscular disease which results in atrophy and paralysis. It is commonly mistaken for chronic anterior poliomyelitis or progressive muscular atrophy, but differs from this in causation, symptoms, and pathology.

Causation.—It is an hereditary affection, or a disease of development or of nutrition. It develops in several children in same family with same or various types.

Pathology.—The lesion is an essential and primary change in the muscle fibres.

1. Individual fibres become hypertrophied.

The nuclei of the muscle fibres are increased in number. The vacuoles appear as evidence of degeneration and fibres split up. At the same time there is an increase in connective tissue about the muscle fibres. Sometimes there is a development of fat between the muscle fibres.

2. Subsequently to the hypertrophy and sometimes without it, atrophy of the muscle fibres occurs with same development of fat and connective tissue or without it.

Appearance during hypertrophy is a very fat limb in which muscles are really weak; during atrophy is a very thin limb in which muscles are very weak or even totally paralyzed.

Symptoms.—In all forms there develops a progressive simultaneous weakness and atrophy of the muscles affected, going

on slowly to complete paralysis. The consequences of such paralysis are evident in size, posture, and deformities, use, motion, and gait. No fibrillary twitching *vs.* progressive muscular atrophy. No R. D. *vs.* chronic anterior poliomyelitis. No sensory disturbance, bladder, or rectal, or trophic skin changes.

Various types of the disease classified by Erb:

I. Infantile muscular dystrophy.

1. Hypertrophic in type.

Pseudo-hypertrophy—fat only.

True hypertrophy, muscle also.

2. Atrophic type.

In limbs only.

In limbs and face also.

II. Adult muscular dystrophy, with similar classes.

I. INFANTILE MUSCULAR DYSTROPHY.—HYPERTROPHIC FORM.

1. *Pseudo-hypertrophic Paralysis.*—Children aged from two to eight affected, males more than females. Only twenty-five per cent. occur after twenty years. Several in one family.

Begins with difficulty in gait. Cannot walk, go up-stairs, or get up. Muscles of back atrophied, and calves of legs hypertrophied. Also infraspinatus and deltoid hypertrophied. Other muscles atrophy, pectorales, latissimus dorsi, triceps, and muscles of thighs.

Posture, shoulders far back. Gait is waddling, and mode of rising by climbing up the legs due to paralysis of extensors of hip is characteristic. Finally, inability to sit up from paralysis of trunk.

After paralysis has occurred shortening of muscles follows, and deformities, talipes equinus, and curvature of spine develop.

No R. D. Knee-jerk normal, then weak. Sensation and sphincters not involved.

Death is due to some intercurrent malady, occurs between twelve and twenty years of age.

Treatment.—Massage. Thyroid extract.

2. *True Hypertrophic Paralysis* (juvenile muscular dystro-

Stress induced decline, [illegible], [illegible]. adult [illegible], but hyper[illegible], [illegible] [illegible] ? [illegible].

phy). Develops in children six to ten. Several in a family. Begins with progressive weakness of muscles.

Muscles affected with hypertrophy are deltoid, infraspinatus, muscles of forearm, sartorius, gastrocnemii.

Muscles which atrophy are: pectorales, latissimus dorsi, serratus magnus, biceps, coracobrachialis, supinator longus, adductors of thighs, glutei and back muscles; finally, calf and peronei.

Gait, posture, and deformities are characteristic. No fibrillary contraction. No R. D. Knee-jerk slowly diminished. No disturbance of sensation or of sphincters.

3. *Atrophic Type in Body and Limbs* (hereditary muscular atrophy). Same features, except entire absence of hypertrophy. Develops from eight to ten years.

4. *Atrophic Type in Limbs and Face* (infantile muscular atrophy; Landouzy-Déjerine type). Face is often first affected.

When face is involved the zygomatic muscles are affected first; loss of naso-labial fold; flat mask-like face; lower lip projects; orbicularis is affected; lips open; labials can't be spoken. Sometimes orbic; palpebræ weak. Facial muscles atrophy slowly.

The other muscles of the body are affected in succession much later, pectorals, latissimus dorsi, biceps, and triceps. Deltoids may be hypertrophied. Back muscles and extensors of thighs are invaded.

Gait, posture, same as in other types.

Any of these types may go on to another type.

II. Adult Muscular Dystrophy.

Any of these forms may occur in adults. Usually a family history can be obtained. Usually develops about age of eighteen, sometimes not till thirty. Some muscles are hypertrophied, others atrophied as in infantile types.

The Central Gray Matter of the Spinal Cord and its Diseases.

The portion of gray matter lying behind the anterior horn, and adjacent to the central canal of the spinal cord and extending

backward to the posterior horn, is made up of gelatinous gray substance and of scattered nerve-cells with a plexus of fibres. It contains one long column of flask-shaped cells—the column of Clarke—in its posterior median part; and an irregular column of small cells in its lateral part—the intermedio-lateral column. The majority of these cells differ from the cells of the anterior horn, in not having a long axis-cylinder process which enters a nerve. They have numerous branching processes, some sending a long branch to the other side through the anterior commissure, others sending a branch out into the antero-lateral column where it bifurcates, turning up and down, and in its course gives off collaterals, which enter the gray matter at various levels. These cells thus form an associated system within the cord.

The function of the central gray matter is to preside over, 1, vasomotor tone; 2, nutrition of all parts; 3, visceral activity, including digestion and excretion, and the activities regulated by the sympathetic system.

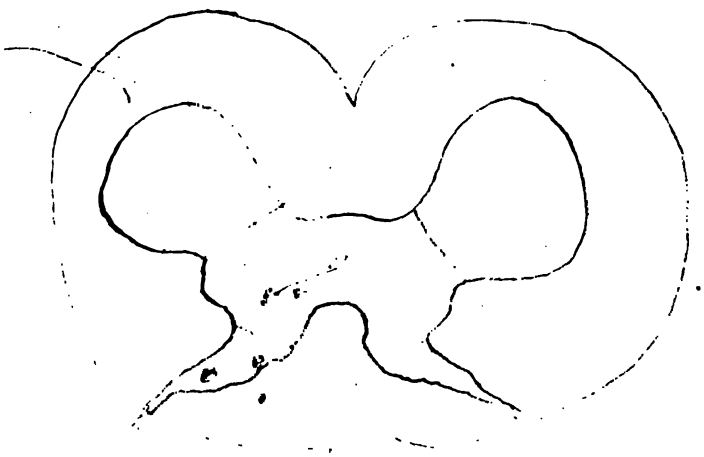
1. Vasomotor innervation is a reflex matter requiring centres with sensory and motor-nerves. Gaskell has proven that these lie in the central gray matter and are joined by fine nerve-fibres which pass in the nerve-roots through the two horns of the cord. The regulation of the flow of blood in accordance with the need of an organ is governed here.

2. The proper degree of metabolism necessary for nutrition in the skin, bone, and joints and muscles, or for repair of tissue, is under the control of nerves whose centres are in the central gray matter.

3. The sympathetic system of nerves presides directly over digestion, circulation, secretion, and excretion, but is in turn controlled by spinal centres which lie in the entire length of the spinal cord in the central gray matter.

The central gray matter receives sensory impulses of pain and temperature sense, and transmits these for some distance on their way upward after they reach it through the posterior nerve-roots; each segment of the cord being related to its own part of the surface, as shown in the table.

All these functions are disturbed in diseases of the central gray matter.



This may be joined with ant. polyomyelitis & If it extends to ant. horn, may have also a progressive muscular atrophy. Cavity may extend up to medulla - then rupture & sudden death.

Syringomyelitis.

Pathology.—This is a rare disease, usually of congenital origin, characterized by a development of gliomatous tissue in the gray matter around the central canal, which subsequently extends outward, involving the entire central gray matter, and then degenerates and is absorbed, leaving a cavity in the spinal cord. Such a cavity may extend through a part of the cord, or through its entire length; varying in size at different segments and thus destroying more or less of the gray matter. Its usual situation is in the the lower cervical and upper dorsal regions. The disease may invade the anterior horns of the cord, causing the symptoms of chronic anterior poliomyelitis. It may invade the posterior horns and columns, causing symptoms of posterior sclerosis. Sometimes spinal gliosis develops in adult life and goes on to the formation of a cavity. Sometimes long perforating hemorrhages occur in the cord, and when the clot is absorbed a cavity remains. Sometimes local softening in the cord leads to the formation of a cavity. Any disease destroying the central gray matter of the cord causes the symptoms of syringomyelitis.

Symptoms.—1. Loss of the sensations of temperature and pain while the sensations of touch and location are retained. The distribution of this sensory disturbance depends on the location of the disease, *e. g.*, if the left hand and forearm only are thus affected, the disease must be limited to the left side of the cervical region of the cord in its lower third, which receives sensations from these parts. Such disturbance of sensation may be extensive, involving irregular areas of the body. It is never symmetrical on both sides.

2. Vasomotor and trophic symptoms. Cyanosis, œdema, blueness of extremities, with lowering of temperature, ulcers, bullous eruptions, sweating, thickened or glossy skin, defective growth of the nails, great brittleness of the bones leading to repeated fractures, and joint affections, also progressive atrophy of the muscles. These occur usually in one limb only.

3. Disturbance in and loss of control over the action of bladder and rectum, and loss of sexual power, are present when the lumbar region of the spinal cord is invaded.

4. Paralysis of the spinal muscles with curvatures of the spine.

Course of the disease is chronic, with slow progress till the symptoms are developed, often including the symptoms of chronic anterior poliomyelitis. It then remains stationary, but if it advances upward and invades the medulla, death may occur at any time from paralysis of the heart and respiration.

The prognosis is bad and no treatment can stop the course.

When tumors destroy the central gray matter or hemorrhages occur in it, the same terminal symptoms may be expected as in syringomyelitis.

The Posterior Horns of the Cord.

This portion of the cord is made up partly of gelatinous matter which occupies its extremity, and partly of a fine plexus of nerve-fibres, in which small cells are irregularly scattered. The function of the posterior horn is to receive the impulses coming in through the posterior nerve-roots, and to transmit such impulses in various directions. These are the sensations of touch, temperature, pain, pressure, and location, and of the muscular sense.

Each nerve of sensation gives off numerous branches on entering the cord, and finally terminates in a brush-like expansion of nerve-fibrils which surrounds some nerve-cell, or in the plexus of nerve-fibrils which make up a fine felt-like tissue in the posterior horn. Any fibre after entering the posterior nerve-root may pass directly into the posterior horn, or may turn up or down in the white matter near the horn at some level other than that of its entrance. The posterior horns are continuous upward (through the medulla and pons) with the ascending root of the fifth nerve. The diseases of the posterior horn will be considered with diseases of the posterior roots and columns, as they uniformly coincide.

The White Columns of the Spinal Cord and their Diseases.

The gray matter of the spinal cord is surrounded by white tissue made up of anterior and posterior nerve-roots and of nerve-fibres passing up or down the cord for longer or shorter distances. The white columns contain therefore:

1. Fibres joining the skin, joints, muscles, and viscera to the gray matter of the spinal cord and entering the various segments through the anterior and posterior nerve-roots.

2. Fibres connecting the various segments with one another.

3. Fibres joining the various segments with the brain.

The white matter can be divided into different columns by observing the process of development—different columns develop at different periods of fœtal life; or by studying the process of disease—different diseases select different columns.

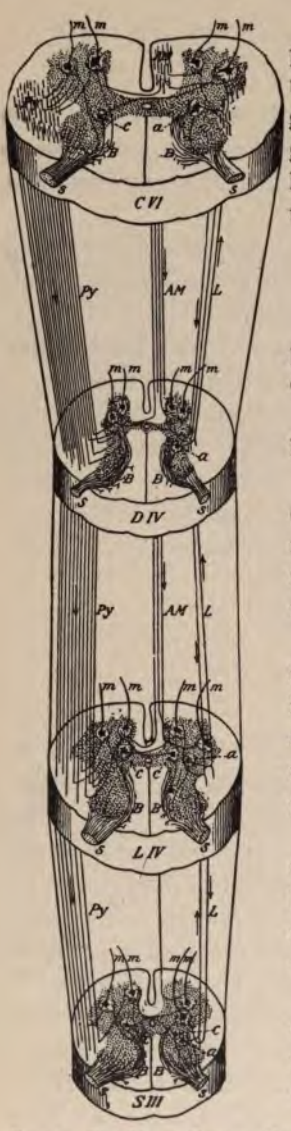
These columns and their diseases are as follows:

The Motor Columns.

1. *Anterior median column.*

2. *Lateral pyramidal column.*

At the lower limit of the medulla each anterior pyramid divides into two parts; one part passes directly on into the anterior median column of the cord; the other part crosses over to the opposite lateral column of the cord, lying near to the gray matter in the angle between the anterior and posterior horn. As the fibres making up these columns pass down the cord, they turn into the anterior gray horns of the different segments—each column becoming smaller the lower it goes. The anterior median column is exhausted at the lower dorsal cord, and therefore does not exist in the lumbar region. The lateral pyramidal column extends to the lowest segment of the cord. These columns transmit motor impulses from the motor cortex of the brain and from the motor ganglia of the brain to the spinal motor centres. These columns also contain fibres whose origin is in the pons and the cerebellum, which pass with the motor fibres from the brain and terminate in the anterior horns of the cord.



They also contain fibres of association passing between various segments of the cord. All these sets of fibres degenerate downward. The area of degeneration is less in extent when the lesion is in the brain than when it is in the cord itself.

Lateral Sclerosis.

Lateral sclerosis is a chronic sclerotic process affecting the lateral pyramidal colums of the spinal cord.

It may be *primary* and is then bilateral.

It may be *secondary* to other diseases in the brain or cord which separate the fibres of the motor tract from their centres of origin and nutrition in the brain cortex. Such diseases are:

1. Tumors, softening or hemorrhage in the brain, destroying the motor tract in one hemisphere, in which case the secondary sclerosis is found in the anterior median column and the opposite pyramidal tract.

2. Congenital maldevelopment of the brain and hydrocephalus, affecting both motor tracts, in which case both anterior median and both lateral columns are sclerotic.

3. Any lesion in the spinal cord, either unilateral or bilateral, or compression of the cord by disease of the vertebræ, in which case the sclerosis

Fig. 8.—The Motor Tracts. *Py*, lateral pyramidal; *AM*, anterior median column, both from right pyramid of medulla to motor neurones of anterior horn, whence motor nerves (*m*) pass to muscles; *s*, sensory nerve entering posterior horn and posterior root zone of the column of Burdach (B), and sending fibres to (*a*) association-neurones; (*c*) commissuralneurones, and also to motor neurones, in anterior horn; *L*, fibres of lateral tract consisting of association-fibres between various segments.

only appears below the level of the lesion, and its extent is greater in area than in 1 or 2

Pathology of Sclerosis.—1. An increase in the thickness of the connective-tissue framework which supports the nerve-fibres. 2. A primary degeneration or a secondary destruction of the axis cylinder in each nerve-fibre, resulting in its swelling, segmentation, fatty degeneration, and final absorption. The result is to reduce the number of fibres in any column of the cord, or even to destroy them entirely, the place of the fibres being filled by connective tissue.

Symptoms of Primary Lateral Sclerosis, or Spastic Paraplegia.—A weakness and stiffness in motion, beginning in the legs and ascending, finally involving the arms. The muscles affected do not atrophy rapidly, as their spinal centres are not diseased. Their reflex and mechanical excitability is increased, as the spinal centres are no longer inhibited by brain impulses. Hence increased knee-jerk and ankle clonus are present. They become rigid from the increased spinal activity. Hence the gait is at first "spastic" (toes drag, knees overlap, motion stiff); finally, when paralysis is complete, the legs are drawn up, knees overlapping and so rigid as to be incapable of passive motion. There are no electric changes in the muscles.

The action of the bladder is imperfectly controlled, and finally involuntary emptying of the bladder at intervals or retention of urine occurs. Constipation is obstinate. In some cases of primary lateral sclerosis the bladder and rectum are not affected. The vascular tone is decreased toward the close of the disease and nutrition is impaired. There are no sensory symptoms.

Course of the Disease.—The symptoms develop slowly and increase until, after many years, permanent paralysis has developed.

Secondary Lateral Sclerosis presents the same symptoms on one or both sides, but the symptoms develop more rapidly according to the nature of the primary disease.

Diagnosis between lateral sclerosis and anterior poliomyelitis:

Lesion in pyramidal tracts.	Lesion in anterior gray horns.
Paralysis usually on both sides equally, in legs, or in legs and arms, never in arms alone.	*Paralysis* may be limited to any single limb, and rarely affects both limbs equally.

All muscles are about equally affected. No muscles are entirely normal.	Certain groups of muscles only are affected. Others escape wholly.
Muscular tone is heightened.	Muscular tone is diminished.
Tendency to rigidity appears.	Muscles are relaxed.
Reflex excitability is increased.	Reflex excitability is lost.
Atrophy is absent, or is slight; and merely due to disuse, hence is gradual in progress. It affects the entire limb.	*Atrophy* is always present in the paralyzed muscles. It advances rapidly, and may become extreme.
Electric contractility is unchanged.	*Electric contractility* is changed. Reaction of degeneration is present within two weeks of the onset.
Vascular tone is diminished; cyanosis and œdema may occur.	*Vascular tone* is diminished, but œdema does not occur.
Paralyzed limb is cold, and sweat may be increased.	Paralyzed limb is cool, but sweat is not increased.
Trophic disturbances in the skin are not infrequent.	*Trophic disturbances* in the skin do not occur.
The control over the bladder and rectum may be diminished or lost.	The control over the bladder and rectum is not impaired.

Treatment.—None. [Read Strümpell on spastic spinal paralysis and on compression myelitis.]

Amyotrophic Lateral Sclerosis.

This is a chronic disease affecting the entire motor system of the spinal cord and cerebral axis, both the groups of cells in the anterior horns and medulla and pons, and also the lateral pyramidal columns in the cord and in the medulla and pons being affected together. The extent of the sclerosis is greater in the cord than in primary lateral sclerosis, as the white column which lies between the pyramidal tract and the gray matter (the *lateral limiting column*) is also sclerotic in this disease. The changes in the anterior horns are the same as in chronic anterior poliomyelitis.

Symptoms.—The symptoms present features similar to those of chronic anterior poliomyelitis and of extensive secondary lateral sclerosis. Weakness, paresis, and atrophy begin in one or both arms and hands and increase slowly. The muscles of the hands and the extensors of the wrists, the deltoid and triceps, are the muscles chiefly affected. The electric contractility is diminished, and finally reaction of degeneration appears. The muscular irri-

tability and reflexes are much increased. Finally contractures result.

Gradual onset of spastic paraplegia in the legs occurs a few months after the arms are affected. Spastic gait develops with increased reflexes. In the final stage the legs are totally paralyzed. There are no sensory disturbances, and bladder and rectum are usually under good control.

After several years symptoms of bulbar paralysis gradually develop, with paresis and atrophy of face, tongue, and throat, and death occurs from respiratory paralysis. Occasionally the bulbar symptoms appear early.

The course is a slow one. The disease may be arrested, but recovery is impossible.

Diagnosis from myelitis of the cervical region and from syringomyelitis by the absence of sensory symptoms; from anterior poliomyelitis chronica by increased mechanical and reflex excitability in muscles atrophied, and by development of spastic gait.

Treatment.—None.

The Sensory Columns.

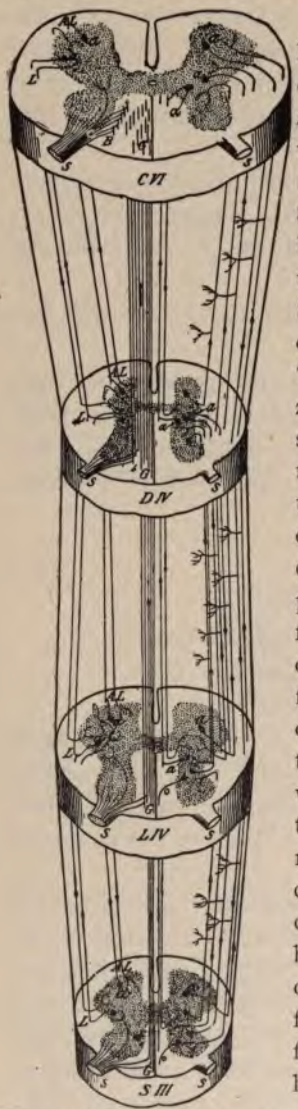
3. *Posterior Lateral Column, or Column of Burdach.*
4. *Posterior Median Column, or Column of Goll.*
5. *Marginal Column, or Column of Lissauer.*

These columns are made up chiefly of sensory fibres which enter the cord in the posterior nerve-roots. They also contain fibres of association between different segments of the cord. Their subdivision will be better understood by a study of the course of the posterior nerve-roots.

The posterior nerve-roots contain fibres which arise from the cells of the posterior spinal ganglia. Each spinal ganglion cell has two branches, or two divisions of one branch; one going out to the surface, the sensory nerve; the other going in to the cord, the posterior nerve-root. The cell in the ganglion nourishes both branches, and if either branch is separated from its cell it degenerates in its entire length. The sensory cranial nerves (X., IX., V.) arise from similar ganglia (jugulare, Gasserian).

The posterior nerve-root enters the cord opposite to the apex

of the posterior horn and then divides into two bundles, a median bundle and a lateral bundle.



(1) The median bundle containing large fibres enters the column of Burdach, forming its root-zone or *lateral zone*. Here a few fibres turn downward into the segment next below (forming the comma-shaped bundle of Schultze) and end in the posterior horn; but the large majority of fibres turn upward to go to higher segments. Some of these soon enter the inner side of the posterior horn (short fibres). These lie near the horn in the lateral zone. Others pass up through several segments (medium fibres). These lie nearer to the median line and in the upper cord are adjacent to the column of Goll, forming the *middle zone* of the column of Burdach. The remainder pass all the way up to the medulla (long fibres), lying at first in the *hinder zone* of the column of Burdach and later forming the column of Goll. For as each root, from below upward, enters the cord, it displaces inward and backward the fibres already ascending; so that the higher the level the larger the number of ascending fibres. In a cross-section of the cord in the upper cervical region the long fibres which have come from the lumbo-sacral region occupy the column of Goll, the sacral fibres lying behind the lumbar; the long fibres from the dorsal region occupy the hinder zone of the column of Burdach;

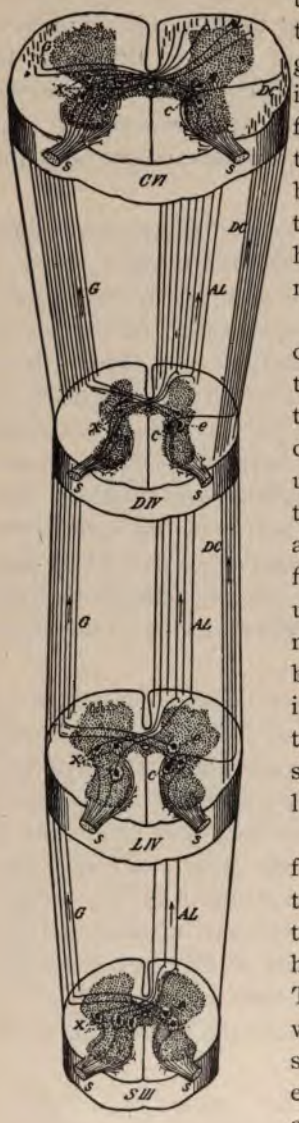
FIG. 9.—The Columns of Burdach and Goll are shown on the left side of the cord. The association-fibres of the cord joining the various segments with each other of various lengths are shown passing through all the columns. The oval column of Flechsig is shown in the lumbar and sacral regions, also called the descending septo-marginal tract.

the long fibres from the cervical region occupy the middle zone of the column of Burdach. It is evident that there is no physiological distinction between the columns of Goll and Burdach. In the anterior part of both columns near the gray commissure lie association fibres and commissural fibres. It is thus evident that the median bundle of posterior nerve-root fibres constitutes the columns of Burdach and of Goll.

The final termination of these fibres is as follows: Each fibre on entering the cord bifurcates, a short branch turns down, a long branch turns up. The long branch in its course upward gives off collateral branches which pass into the gray matter of the posterior horn, and finally the branch itself ends there. In all cases the termination is by means of a brush-like expansion of nerve-fibrils. The short fibres end in the fine network making up the substantia gelatinosa of Rolando in the posterior horn, and about the cells of the posterior and central gray matter. The medium fibres end in a network which surrounds the cells of the column of Clarke in the central gray matter. A few fibres pass forward to the anterior horn and to the opposite side through the posterior gray commissure. The long fibres pass up to the nuclei gracilis and cuneatus in the medulla.

(2) The lateral bundle of the posterior nerve-root consists of both large and small nerve-fibres. Many of these pass directly into the gray matter of the posterior horn, where some end in a network of fibres about the cells of the gelatinous substance, or deeper in the horn; and others pass forward and cross over in the gray commissure to the opposite side, where they turn outward into the antero-lateral column or backward into the column of Goll. The smaller fibres bifurcate on entering the cord and turn down and up, forming the column of Lissauer, in which they pass but a short distance and then turn into the horn. This column is of uniform size through the cord, and hence cannot contain any long fibres.

6. *Antero-lateral Ascending Tract or Column of Gowers.*— In the antero-lateral column of the cord lying on the periphery just in front of the direct cerebellar column is a small tract which ascends through the cord and enters the formatio reticularis of



the medulla, where it appears to end in the gray cells of this formation. It begins in the lumbar region, and increases in size as it ascends, receiving fibres from the cells of the central gray matter. It degenerates upward. It has been supposed to convey sensations of temperature and pain upward after they have passed through the central gray matter.

7. *Direct Cerebellar Column.*—This column lies on the lateral periphery of the cord just outside of the pyramidal tract and behind the antero-lateral ascending tract. It is very small in the upper lumbar region, becomes larger in the dorsal region, and is of considerable size in the cervical region. Its fibres come from the cells of the columns of Clarke in the central gray matter. It passes into the restiform body in the medulla and through this into the cerebellum, where it ends in the vermiform or middle lobe. It is supposed to convey sensations of equilibrium in the act of walking.

It is evident that the sensory fibres from the posterior nerve-roots are distributed to many different columns of the spinal cord, and even in the medulla have numerous different connections. This anatomical fact may be associated with the physiological fact that one sensation is capable of producing several different effects, *e. g.*, a burn causes a reflex withdrawal of the part burned,

FIG. 10.—On the right side the termination of Sensory Nerves about the Cells of Clarke's Column (*C*) and about the Intrinsic Cells (*e*) is shown. From *C* fibres pass up in *D C*, the direct cerebellar column. From *e* fibres pass over to the column of Gowers (*G*). On the left side the termination of sensory fibres about intrinsic cells (*x*) is shown. From *x* fibres cross to the antero-lateral column and ascend.

vascular changes, and a process of repair, sensations of heat, touch, and pain, accurately localized, followed by conscious effort to protect the part: *e. g.*, stumbling in the act of walking causes reflex balancing movements involving motions of the entire trunk and arms to reëstablish the centre of gravity or protect against fall, also conscious sensation of unstable position, possibly vertigo, quickened respiration, action of ocular muscles and head to observe danger, sensation of contact, possibly pain, in the toes, and corresponding vascular changes locally, and finally voluntary effort to correct position after an appreciation of all the various sensations received.

We distinguish conscious sensations of contact, temperature, pain, muscular sense, including pressure and position, and unconscious sensations, which set up reflex and automatic acts, vascular changes, variation in thermic and nutritive processes, and changes in muscular tone. These all enter by the posterior nerve-roots, and set up activities in the central nervous system.

Locomotor Ataxia.

This is a chronic disease of the sensory part of the nervous system, the posterior nerve-roots and the posterior columns of the cord being diseased. It is also called *tabes dorsalis*, or *posterior spinal sclerosis*.

Pathology.—There is a degenerative neuritis of the posterior nerve-roots, and sometimes of the sensory part of the peripheral nerves. There is a sclerosis of the posterior columns of the spinal cord, beginning in the lateral or root-zone of the column of Burdach and in the column of Lissauer, extending thence to the middle zone of the column of Burdach, then to the column of Goll, and finally to the hinder zone of the column of Burdach. Thus both posterior columns are eventually diseased in their entire extent. The disease usually begins in the lumbo-sacral region and extends upward through the cord. It is many years before it invades the cervical region or destroys the entire area of the posterior columns. The terminal network of nerve-fibres in the posterior gray matter, and especially about the column of Clarke, is destroyed by the disease.

Theories of the disease: (1) It is a primary sclerosis of the posterior columns of the cord. (2) It is a sclerosis of the root-zone, due to disease of the vessels entering this zone. (3) It is a primary disease of the cells of the posterior spinal ganglia, with secondary degeneration of the sensory fibres entering the cord from the ganglia, and compensatory sclerosis in their course.

Etiology.—It is a disease of adult life, persons under twenty-five rarely being affected, and it is rare among women. The chief predisposing cause is syphilis, which precedes it in seventy per cent. of the cases. The exciting causes are great physical exertion, exposure to cold and wet, and alcoholic and sexual excesses.

Symptoms.—The symptoms of the disease are very numerous, but appear in succession. They may be classified according to the time of their appearance. There is a stage of pain, a stage of ataxia, and a stage of paralysis.

Symptoms in Stage of Pain.

Paræsthesiæ. Numbness, formication, sensation of dead extremities, cotton or pins in soles of feet and fingers, coldness, itching of anus and scrotum or of other parts.

Pain. Short, sharp, cutting, boring. Deep pain; comes in attacks, one second to one-half minute repeated, or in severe attacks for several hours or days with intermissions, rarely continuous attacks. Begins in thighs and legs. Pain is a prominent symptom early, but may continue through all stages. It may be mistaken for rheumatism or sciatica, but its character is different. It is the same as in alcoholic multiple neuritis, but there tenderness exists. There is no relation between intensity of pain and degree of ataxia. As disease advances upward the pains extend. Pain may be absent or insignificant in some cases, especially in cases beginning with optic-nerve atrophy.

Pain in the small of the back and loins of an aching character may occur.

Girdle sensation or sensation of tightness and pressure intense and severe, about the legs and body. This ascends gradually as the disease advances.

Loss of knee-jerk, called Westphal's symptom.

Imperfect control of bladder. Slow urination. Slight drib-

bling, and hasty urination. Finally control is very imperfect and may be painful. This may be a dangerous complication, since death from cystitis occurs.

Impotence; occasionally priapism and sexual excitement precede this.

Constipation; usually obstinate throughout.

Loss of pupil reflex to light, the reaction in accommodation being preserved—Argyle-Robertson pupil.

Contracted pupil, "myosis spinalis," is frequent but not constant.

Symptoms in Stage of Ataxia.

In addition to those of first stage there develop:

Anæsthesia and analgesia. Sense of pain impaired and delayed. This may be tested with faradic brush. It prevents injuries from being perceived; hence ulcer of foot and Charcot joints occur as complications. Hence, guard against accidents in giving hot baths, blisters, etc.

Hyperalgesia. Sensation of pain may be produced by any touch, and pain is felt severely. This occurs in a few cases early in the disease.

Change in temperature sensation. Cold felt keenly and heat less than normal.

Complete anæsthesia develops late in the disease and in the extremities.

Hypotonia; an undue mobility of joints from lack of tone in muscles.

Impairment of muscular sense. Muscular sense is the sense by which the situation and movements of the limbs are appreciated, and pressure and weight are felt. Not same as effort sense. Imperfect perception of weight and pressure is a symptom observed late. Situation of limbs is imperfectly perceived. Hence, one means of perfect guidance of limbs is removed. Hence, patient walks badly in the dark, and cannot touch objects accurately with eyes closed; hence he watches his own motions—aiding his muscular sense by sight. Therefore a blind ataxia is the most helpless of all.

Ataxia is imperfect co-ordination of muscular action. For

every compound act a regular succession and proper degree of motions are needed, *e.g.*, closure of fist, walking. This regulation is largely automatic and is carried on in subcortical centres, which receive unconscious sensations through the posterior nerve-roots. It may be voluntary, by an effort of attention, and is so at first, for all motion, and always for highly complex motion, *e.g.*, writing. Hence ataxia of movement may occur in many diseases. It is a prominent symptom in locomotor ataxia.

Romberg's symptom—swaying when standing with eyes closed.

Ataxic gait—legs wide apart, feet lifted too high, and planted too forcibly, steps being of irregular length and body imperfectly balanced.

Imperfect use of hands in dressing, writing, etc. Irregular contraction of muscles on effort. Imperfect muscular tone.

Tests of ataxia. Stand with eyes closed, walk, turn suddenly or walk backward; touch heel to toe, or heel to knee; touch finger to nose or both fingers with eyes closed; pick up pin; button clothes.

Symptoms occasionally present, due to complication of neuritis in various nerves. Optic-nerve atrophy, with progressive blindness, develops in ten per cent. of the cases. Optic disc looks white, then gray. Arteries appear small and walls thin. Visual field is contracted for colors and for light. Finally total blindness occurs.

Paralysis of the ocular muscles.

VI. n. paralysis; convergent strabismus, with contracted pupil.

III. n. paralysis; divergent strabismus, with dilated pupil and ptosis. Nystagmus rarely occurs.

Deafness develops in a few cases from atrophy of auditory nerve.

Crises—(1) gastric; sudden vomiting with pain, for several hours, even for days. Great prostration follows. Rectal feeding necessary. Intestinal and rectal crises with diarrhœa and tenesmus have been observed. (2) Laryngeal; sudden and severe cough, with spasm of larynx and suffocation. (3) Cardiac; attacks of

angina pectoris. All due to a complicating peripheral neuritis in pneumo-gastric nerve. These symptoms are quite exceptional.

Trophic disturbances are more common in stage of ataxia.

Charcot joints. Knee, elbow, ankle, wrist. Enormous swelling with effusion without pain. Erosion of ends of bones and destruction of articulation. Due to injury.

Perforating ulcer of foot, caused by a corn, or a subcutaneous hemorrhage, or a caries of bone neglected.

Rarefication of the bones with production of fractures.

Irregular muscular atrophies develop late in the disease.

Herpetic eruptions and pemphigus.

These are due to a combination of causes, chiefly to a loss of pain sense which prevents injuries from being perceived, or to a complicating peripheral neuritis.

Symptoms in Stage of Paralysis.

General nutrition remains good, but ataxia becomes so extreme as to render patient helpless and confine him to bed.

Control of bladder completely lost, and urine dribbles constantly.

Accidents produce injuries, and trophic disturbances follow.

Hence patients die of bedsores, cystitis, exhaustion, or of complicating penumonia.

Course of the disease is chronic through the three stages, lasting twenty to forty years.

Usual mode of onset is with pain, loss of knee-jerk, bladder trouble, impotence, and ataxia.

Occasional modes of onset: (1) blindness, loss of knee-jerk, imperfect gait, numbness, but no pain.

(2) Gastric crises at intervals, loss of knee-jerk, then pain and ataxia.

(3) Various forms of strabismus, myosis, then ataxia of arms.

There are remissions in the symptoms and complications may subside, but no true arrest occurs or recovery, after the stage of ataxia has begun.

Diagnosis. Early, by means of pains, fatigue, loss of knee-jerk, and Argyle-Robertson pupil. Late, by ataxia and bladder disturbance.

Diagnosis from multiple neuritis. See page 11.

Treatment. Moderate exercise, avoiding all fatigue. Good general diet without special restrictions, which weaken patient. Spinal douches, tepid or cool, never extreme. Massage. Practise in fine movements to overcome ataxia. Medicines—arsenic, nitrate of silver, ergot. Anti-syphilitic remedies.

Treatment of pain by antipyrine, etc., gelsemium, opium, faradization, hot applications. Of crises by counter-irritation and morphine. Of optic atrophy by strychnine. Of trophic disturbances by rest and apparatus.

Combined Sclerosis.

This is a Sclerosis of the Lateral and Posterior Columns (*rare*).

(1) Congenital cases. *Friedreich's Hereditary Ataxia* is due to defective development of the lateral and posterior columns. It occurs in several members of a family, and is hereditary.

Symptoms.—As the children grow up they are found to be awkward. They walk badly, and do not use arms well. Reflexes are lost, exceptionally exaggerated. Dull pains in legs, not lightning in character. Deformity in feet. No bladder symptoms, and no trophic changes, and no crises. Ataxia is marked and goes on to actual paralysis. Speech may be thick and nystagmus present. The condition is a chronic one, lasting during life. Children often become imbecile.

(2) Acquired cases. *Ataxic Paraplegia* of Gowers.

Etiology.—Occurs in males chiefly, between thirty and forty years of life. Not after syphilis, but after exposure to cold or sexual excess. Possibly after concussion of spine.

Symptoms.—Slow onset of weakness and ataxia together. Undue fatigue, unsteady gait. Romberg symptom. Dull pain in back and legs, no lightning pains. Reflexes increased. Knee-jerk high, ankle clonus present. Sexual power lost. Sphincters less impaired than in tabes. Eye symptoms very rare. Tremor of face and speech sometimes develops.

Differs from tabes in fact that the nerve-roots are not involved.

Course.—Weakness increases slowly to paralysis. Rigidity soon develops, and contractures. Death occurs from complications.

The Association Columns.

8. *Antero-Lateral Column.*—There is a large collection of nerve-fibres forming a white column lying adjacent to the gray matter of the cord, in front and inside of the pyramidal tract, and inside of the direct cerebellar and antero-lateral ascending tracts, extending as far forward as the anterior nerve-roots. (See Fig. 1.) A portion lying next to the gray matter has been named *the lateral limiting layer.* This column contains a few fibres, which degenerate all the way upward to the medulla, and it is possible that some sensory impulses are transmitted through it. (See Fig. 9.) The major part of the fibres are, however, short, and serve to connect the various segments of the cord with one another. (See Fig. 8.) In it pass the majority of the "association fibres" of the cord, which harmonize the action of different parts.

9. *Anterior Column.*—This lies between the exit of the anterior nerve-roots and the anterior median column. It has the same function as the antero-lateral column, and like it ends in the formatio reticularis of the medulla.

There are very many polygonal cells scattered through the gray matter in both horns and in the central gray which send out to these columns an axis-cylinder process, which on entering the column bifurcates, sending one branch down and the other upward. These branches give off collaterals in their course, which pass into the gray matter at various levels and terminate in brush-like expansions. The original branches terminate in the same way after having a longer or shorter course. Thus the majority of the fibres in these columns are true association fibres. There are a few such fibres also in the portion of the posterior columns adjacent to the gray commissure.

Secondary Degenerations.

When the cord is divided by an injury or by transverse myelitis there results a degeneration in the various columns.

The anterior median and lateral pyramidal columns degenerate downward for their entire length. There is also a short tract of degeneration downward in the lateral zone of the column of Burdach. The anterior and antero-lateral columns degenerate downward for a short distance.

The columns of Burdach and Goll and Gowers and the direct cerebellar column degenerate upward through their entire length. The columns of Lissauer, and the anterior and antero-lateral columns degenerate upward for a short distance.

Such degenerations are due to the fact that the transverse lesion cuts off the individual fibres from their nutrient cells, and hence the distal end degenerates, the fibres nourished by cells above the lesion degenerating downward and those by cells below the lesion degenerating upward.

Myelitis.

This is an inflammation or softening of the spinal cord involving an entire segment, both gray and white matter.

Pathology.—Cord looks normal, but feels soft at points; or may break in two and be soft enough to be fluid. On cutting it the cut surface swells out as if semifluid; the gray matter looks red and capillary hemorrhages are seen; the limit of white and gray is indistinct.

Microscopically.—Large numbers of inflammatory corpuscles everywhere. Nerve-cells and fibres swollen and degenerated, granular and disappearing or atrophied. Neuroglia cells increased. Deiter's spider cells present. Fatty and granular cells everywhere as evidence of degeneration. Vessels are distended with blood and ruptured; walls may be infiltrated and thickened.

Secondary degenerations as already enumerated.

Etiology.—Males more than females. Soldiers, porters. Age, ten to forty. Exposure to cold. Overexertion. Falls and blows. Concussion of cord. Sexual excesses. Menstrual suppression. After confinement. Injuries of the cord. Compression of the cord by disease of spinal bones or tumors. Secondary to acute infectious diseases. Syphilis and chronic alcoholism,

often due to homorrhage or to thrombosis in diseased vessels, "spinal apoplexy."

Varieties, General.—Entire cord affected—ascending or descending.

Disseminated.—Various segments at different levels affected.

Transverse.—One or two segments at one level affected.

Symptoms.—(1) Direct, due to destruction of cord tissue.

1. Motor. Paralysis, atrophy. R. D. Loss of tone in muscles.

2. Reflex. Loss of spinal reflexes. Paralysis of the bladder and rectum, impotence

3. Sensory. Numbness. Pain. Paræsthesia, anæthesia. Hyperæsthesia at upper level of lesion. Pain and tenderness of back to heat.

4. Vasomotor and trophic. Imperfect circulation. Skin cold. Sweat profuse. Bedsores on buttocks, sacrum, and heels.

The distribution of the direct symptoms depends on the extent of the lesion. If general they are universal. If disseminated they are scattered. If transverse they are limited to one level.

Diagnosis of level from situation of symptoms, motor or sensory, see table, page 14.

Symptoms.—(2) Indirect, due to cutting off of impulses to other parts. The cord transmits impulses to and from the brain. In disseminated and transverse myelitis tracts are broken. (1) Motor tracts to parts below, hence secondary lateral sclerosis with its symptoms. (2) Sensory tracts from parts below, hence secondary degenerations upward with their symptoms.

Secondary symptoms are:

1. Rigidity and increased reflexes, contractures, spasms of legs, paralyzed muscles not atrophied, and no R. D.

2. Loss of bladder control without bladder paralysis, priapism.

3. Imperfect sensation without incoördination.

4. Imperfect perception of pain; hence bedsores from dirt, bedsores over sacrum, glutei, heels, and ankles.

Examples: In general myelitis, direct symptoms throughout body.

In disseminated myelitis, direct symptoms in irregular areas, few indirect symptoms.

In transverse myelitis, direct symptoms very limited, indirect symptoms below the lesion.

(*a*) Cervical transverse myelitis, direct symptoms in arms, indirect in legs.

(*b*) Dorsal transvere myelitis, direct symptoms few, in trunk, indirect in legs like lateral sclerosis, arms free.

(*c*) Lumbar transverse myelitis, direct symptoms in legs, arms free, no indirect symptoms.

Course of General Myelitis and Disseminated Myelitis.—Acute or subacute, onset in two weeks after a cold or great strain or after typhus, typhoid, small-pox, confinement, syphilis. Weakness in limbs. Pains in back and limbs. Prostration. Then paralysis. Pain and anæsthesia. Bladder and rectal symptoms. Then patient is confined to bed and bedsores develop. Slow course for a year to three years and occasionally gradual, imperfect recovery. Usually cystitis, bedsores, or pneumonia and death.

Course of Transverse Myelitis.—Acute, onset after injury—blow, fall, wound, dislocation of spine, or strain, causing hemorrhage in cord; or from embolism of spinal arteries.

Chronic, onset after spinal caries with deformity and pressure, or tumor in spinal canal or cord. Course depends on possibility of removing the cause.

Diagnosis.—Myelitis *vs.* multiple neuritis, see page 11.

MYELITIS	*vs.*	MENINGITIS.
Very rapid onset.		Slower onset.
Fever moderate.		Fever high.
Pain in back moderate, in limbs or body dull.		Pain in back and body and limbs very severe, increased by motion.
Hyperæsthesia slight in a band at upper level of lesion.		Hyperæsthesia of entire body and limbs very intense.
Anæsthesia below lesion.		No anæsthesia.
No spasms or rigidity.		Spasms of limbs and back with rigidity of back appear early.
Paralysis total in some muscles with atrophy and R. D.		Paralysis only apparent; due to fear of pain on motion, no atrophy.
Sphincters paralyzed.		Sphincters not affected.
Bedsores and cystitis.		No bedsores or cystitis.

Myelitis	*vs.*	Hysterical Paraplegia.
Anæsthesia below lesion includes genitals.		Anæsthesia varies in level and genitals never included.
If thighs are paralyzed and atrophied reflexes are lost.		When legs are paralyzed reflexes are exaggerated.
Usually some muscles atrophy and show R. D.		Muscles rarely atrophy and R. D. never appears.
Sphincters usually involved.		Sphincters rarely involved.

Diagnosis of level of lesion by distribution of anæsthesia, see table, page 14.

Treatment of myelitis.

When onset is acute remove cause, and use counter-irritation, ice-bag, or warm douche. Keep at perfect rest in prone position. Give sedatives and ergot; in early stage give purgatives, calomel, or salts.

When case is chronic in onset or in course: employ counter-irritation. Cups, blisters, cautery, ether spray; but not in cases with tendency to bedsores. Use baths, douches, tepid and cold, to spine. Keep up strength of patient by good diet, and regulate digestion. Use massage to the limbs—hot baths for rigidity. Use interrupted galvanic current to atrophied and paralyzed muscles.

Prevent cystitis by keeping catheters and parts clean and aseptic.

Treat cystitis by washing out the bladder, as in surgery.

Prevent bedsores by careful padding or water-bed, by sponging with alcohol and alum-water, by frequent change of position.

Treat bedsores by aseptic dressings, as in surgery.

For pain, apply heat or faradic brush to part. Give antipyrin, antifebrin, phenacetine, morphine, salicin, salicylic acid.

For spasms, apply heat to spine, or hot bath to legs, or cups. Give bromides. For rigidity, when beginning, pads and massage.

For incontinence of urine, pads of absorbent cotton, or wear urinal.

Medicines in chronic myelitis: Iodide of potash, gr. x. to xx., t.i.d. Nitrate of silver in pill with kaolin, gr. 1-4 t.i.d. increased. Ergotin when congestion is suspected. Strychnine when an increase of reflex activity is not present. Phosphorus and arsenic as nerve tonics. Use ung. hydrarg. and KI in large doses in syphilitic cases.

Hemorrhage into the Spinal Cord.

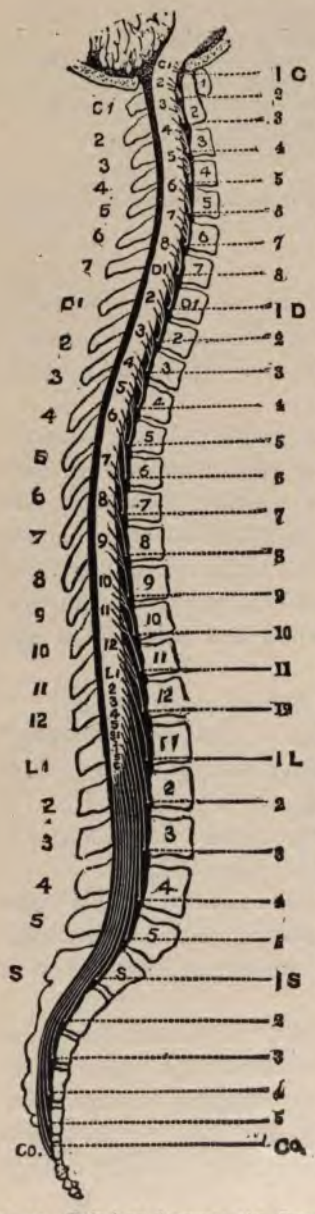


Fig. 11.—Relations between the Segments of the Spinal Cord and their Nerves and the Bodies of the Vertebræ. (Gowers.)

Hemorrhage into the spinal cord or into the membranes is a rare occurrence, and is almost always due to traumatism. The symptoms produced are those of sudden suspension of all spinal functions at and below the level of the hemorrhage. They are therefore identical with those of transverse myelitis, and the sudden onset affords the only means of distinguishing the two diseases. Sometimes spontaneous hemorrhages in the auterior horn cause symptoms of anterior poliomyelitis. Sometimes central hemorrhages cause symptoms of syringomyelitis.

Tumors of the Spinal Cord.

Tumors rarely grow in the spinal cord. They are usually secondary to tumors elsewhere. All varieties may occur. The symptoms produced are those of transverse myelitis of slow onset and characterized by extreme pain at the level of the tumor or in the sensory areas which correspond to the segment in which the tumor grows. The only relief is by operation. The relation of the segments of the cord to the spinal column is shown in Fig. 11.

Brown-Séquard's Paralysis.

Hemorrhages in the cord, tumors in or about the cord, and bony growths or callus, the result of injury, sometimes divide or com-

press one-half only of the cord. The symptoms resulting were first described by Brown-Séquard, and hence the condition has been named after him. This condition is as follows: (1) on the side of the lesion there is paralysis with increased reflexes, and sometimes a loss of muscular sense below the level affected, and a marked hyperæsthesia to touch and pain; (2) on the side opposite to the lesion there is no paralysis, but there is a loss of sensation of touch, temperature, and pain, and sometimes a development of bedsores below the level of the lesion; (3) at the level of the lesion there is a band about the body of anæsthesia ,with another band of hyperæsthesia above it; the band on the side of the lesion being a little higher than upon the opposite side.

THE BRAIN AND ITS DISEASES.

I. The Cortex of the Cerebral Hemispheres.

The cortex consists of gray matter spread out in a layer over the cerebral hemispheres, 3mm. thick and about 200,000 sq. mm. in extent; its amount varying greatly in different persons; its development determining mental capacity. The development of fissures throwing the cortex into folds enables this large extent to be contained in the skull.

The Microscopic Appearance of the Cortex.—Cells arranged in layers.

(1) Superficial or molecular layer $\frac{1}{10}$ small cells. Much neuroglia.

(2) Layer of small pyramids $\frac{1}{10}$ with many branches and short process.

(3) Layer of large pyramids $\frac{4}{10}$ with few branches and a long projection-fibre process.

(4) Layer of polymorphous cells $\frac{2}{10}$ with many short branches and a short process.

(5) Layer of fusiform cells $\frac{2}{10}$ with short branches and association-fibre process.

Fibres trasverse these layers, giving off collaterals, and form an intricate network throughout the gray matter. There are great variations in the structure of different regions. (See "Quain's Anatomy," 10th Ed., Vol. III. Starr's "Atlas of Nerve Cells.")

The fibres arising from the cortex and constituting the white matter beneath it are (1) association fibres passing to some other part of the cortex; (2) commissural fibres passing to the cortex of the opposite hemisphere; (3) projection fibres passing to the basal ganglia, cerebral axis, or spinal cord.

Divisions of the Cortex into Lobes and Convolutions.—

Frontal lobe, 40 per cent.; parietal lobe, 20 per cent.; temporal lobe, 20 per cent.; occipital lobe, 17 per cent.; Island of Reil, 3 per cent. of extent of cortex.

Landmarks on the Cortex.—Fissure of Sylvius, fissure of Rolando, interparietal fissure, parieto-occipital sulcus, calcarine fissure, callosomarginal fissure.

Cranio-cerebral Topography.

To find the fissure of Rolando, lay down a line from the root of the nose to the occipital protuberance over the top of the head and take a point 0.557 of the distance back upon this line. This

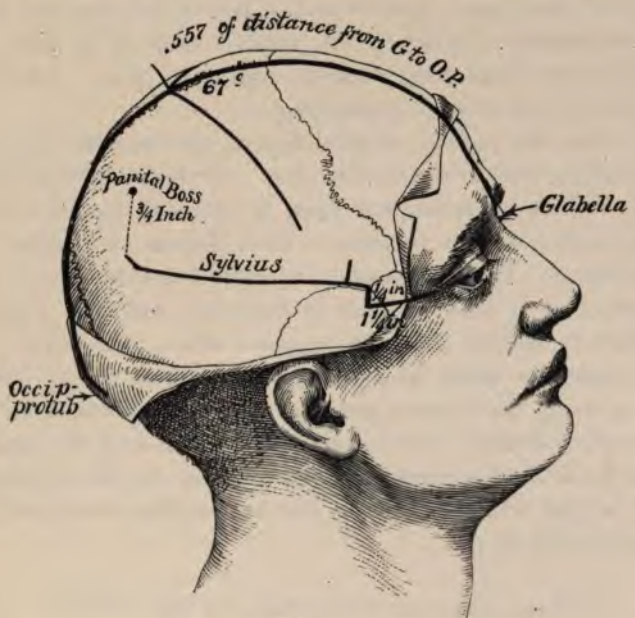


Fig. 12.

point will correspond to the upper end of the fissure. The fissure makes an angle of 67° with the median line just measured. Hence if two strips of metal, fixed to one another at this angle, be placed on the head with their junction upon the upper end of the fissure, when one strip is on the median line the other strip, pointing forward and downward, must lie over the fissure of

Rolando. In its lower third the fissure becomes a little more vertical than the strip. The fissure is about three and a half inches long.

To find the fissure of Sylvius, lay down a base line from the lower margin of the orbit to the auditory meatus. Lay down a second line parallel to the base line from the external angular process of the frontal bone backward one inch and a quarter, and then measure upward one-quarter of an inch; this gives point one. Fnd the most prominent part of the parietal eminence and from it draw a line downward perpendicular to the base line, and on this take a point three-quarters of an inch below the eminence; this gives point two. Join these two points, and the line will lie over the fissure of Sylvius. The anterior limb of the fissure will be two inches behind the external angular process. The fissure of Sylvius is about four inches long.

To find the parieto-occipital fissure, continue the line of the fissure of Sylvius to the median line. At their junction lies this fissure. Since all areas now open to surgical operation can be located with a definite relation to these fissures, no further rules are necessary. Since in opening the skull it is customary to make a fenestra of at least an inch in diameter, and it is frequently necessary to enlarge the opening much more, a procedure in no way dangerous under aseptic conditions, there is no difficulty in recognizing the fissures and convolutions exposed if the rules are closely followed. Prior to the large incision of the scalp it is well to mark certain points upon the skull by the sharp point of a chisel, so that when the bone is laid bare surface landmarks may still be kept in view.

Localization of Functions in the Cortex.

Proofs from comparative anatomy; natural and experimental atrophy; process of development; irritation and excision of cortex with various results; pathological lesions of limited extent with secondary degenerations.

The functions of the cortex in general are:

(1) To receive impressions from the various sensory organs as conscious perceptions.

(2) To associate these various simultaneous perceptions into a mental image or concept.

(3) To retain these concepts as memories so that objects can be recognized or recalled.

(4) To give expression to thought in action and in speech.

(5) To restrain the flow of thought and to inhibit or control automatic and reflex acts.

It has been established that various powers of perception, memory, and volition can be definitely assigned to various parts of the brain; that each sense has its corresponding area in the cortex, and each voluntary movement its point of departure from the cortex.

It has been proven that perceptions leave behind them a physical trace inseparably connected with the area in which they were originally received; that irritation of this area produces a renewal in consciousness of the original perception as a memory, which may be so vivid as to appear real, being then an hallucination; that destruction of this area causes a loss of the power of recalling the previous perception or of recognizing it when repeated.

It has been proven that movements which are acquired by practice, and are therefore the result of conscious effort, are always initiated from a definite area; that this area is inseparably related to movements; since its irritation, by any cause, results in the production of forcible movements of the nature of spasms, and its destruction produces a loss of the power of movement or paralysis of voluntary action.

It has been proven that the use of language involves both the recollection of auditory and visual symbols and the initiation of impulses of speech and writing, thus calling into play not only the motor portion of the brain, destruction of which therefore causes a loss of speech, but also the sensory portion of the brain, a lesion of which will also cause aphasia.

There are certain areas upon the cortex of the brain, not necessarily coextensive with either lobes or convolutions, whose functions are accurately known.

These areas are: (1) The sensori-motor area. (2) The visual area. (3) The auditory area. (4) The area of sensations of smell and of taste. (5) The speech areas.

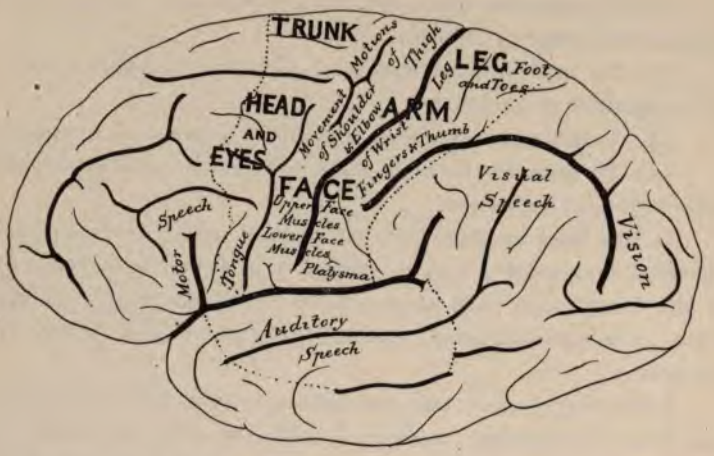


FIG. 13.—Diagram of the Fissures and Convolutions of the Convexity of the Left Hemisphere of the Brain, with the areas presiding over various functions. The speech areas are shown on this hemisphere. The motor area is more extensive on the left than on the right hemisphere.

THE SENSORI-MOTOR AREA.

The sensori-motor area includes the cortex of the anterior and posterior central convolutions which border the fissure of Rolando and the adjacent cortex in front and behind these convolutions. Each hemisphere controls movement on the opposite side of the body, but as the right hand is more generally used and is better trained than the left, this area is larger on the left hemisphere than on the right.

The cortex of the posterior part of the second frontal convolution controls the movements of the eyes and head. Impulses starting from this area produce conjugate movement of these parts toward the opposite side. The eye district is below, the head district above.

The lower third of the anterior and posterior central convolutions governs the movements of the face, tongue, larynx, and pharynx. The eyebrows and cheeks are controlled by the upper and forward part of this area; the tongue and larynx by the lower

and forward part; the mouth, pharynx, and platysma by the hinder part.

The middle third of the anterior and posterior central convolutions governs the movements of the upper extremity; the shoulder motions being controlled in the anterior and upper part of this area, the elbow motions in its middle part, and the hand and finger motions in its posterior and lower part.

The upper third of the anterior and posterior central convolutions, including their junction in the paracentral lobule, controls the motions of the lower extremity, the thigh, knee, foot, and toes being governed by various parts of this area from before backward in the order named.

The parts susceptible of the finest and most delicate movements, those directed by the most acute sensations, the lips, the fingers, and the toes, lie furthest back in the motor area, in the depth of the fissure of Rolando and in the posterior central convolution. Lesions in this convolution almost always cause some loss of tactile sensation as well as paralysis, and hence this area is thought to be the seat of tactile sensations as well of movements, while some cases point to the localization of muscular sensations in the area just behind that of motion.

The trunk or body motor centres lie between those of the leg and arm.

There are no sharply defined sections of the motor area to be assigned to special motions. Each motion, each part of a limb, has a wide general representation over the cortex and a special representation at a limited area. The areas of representation of different limbs merge into one another; thus, in the representation of the thumb we find that there is a focus, but that the thumb is represented over a great deal of the upper limb region, and that this representation diminishes in intensity gradually as we pass from the focus upward. This explains the fact that the excision of a small area does not totally paralyze the portion of the limb represented chiefly on that area. The adjacent areas represent to some extent that limb, and hence can govern it if need be.

The motor centres govern motor acts rather than special muscles, each act involving a succession of movements of different joints by various groups of muscles.

Cortical Spasm and Paralysis.—Irritation in the motor area produced by any kind of disease gives rise to localized spasms or convulsions, called Jacksonian epilepsy. Destruction of the motor area gives ries to paralysis. The point of beginning of the spasm or the extent of paralysis depends on the part of the motor area diseased. Hence, from the character of either of these symptoms a conclusion can be reached as to the location of the disease. Cortical spasm or paralysis usually begins in the face or in one limb of one side and extends. The order of the progress also indicates the location of the disease, *e. g.,* from face to arm, then to leg; or *vice versa, e. g.,* spasm of shoulder, arm, and hand, *vs.* spasm of fingers, arm, and shoulder: the disease is most intense in the part first affected. Sensations of numbness precede the spasm. A cortical spasm begins locally, and usually extends until one side of the body is affected. It is usually followed by temporary weakness. Consciousness is preserved.

Cortical paralysis is monoplegic in type *vs.* subcortical paralysis, which is hemiplegic in type. A slowly advancing paralysis indicates a slowly extending destruction of tissue. Cortical paralysis is associated with increased reflexes, but there is no atrophy and no change in electric reactions. Contractures develop late.

The Visual Area.

The area of sensations of sight is located in the occipital lobe of the brain, including the cuneus on the median surface and the occipital convolutions on the convexity. The cortex lying in the calcarine fissure is the part primarily reached by the visual impulses, but the parts named are also concerned in vision. Each occipital lobe receives impressions from one-half of both eyes; hence a lesion in one lobe produces hemianopsia, a half-blindness in both eyes, the blind field of vision being on the opposite side to the lesion. This is termed homonymous hemianopsia; that is, a blindness in the like-named halves of both visual fields. When the right occipital lobe is affected, the patient cannot see anything on the left side of the middle line as he looks forward.

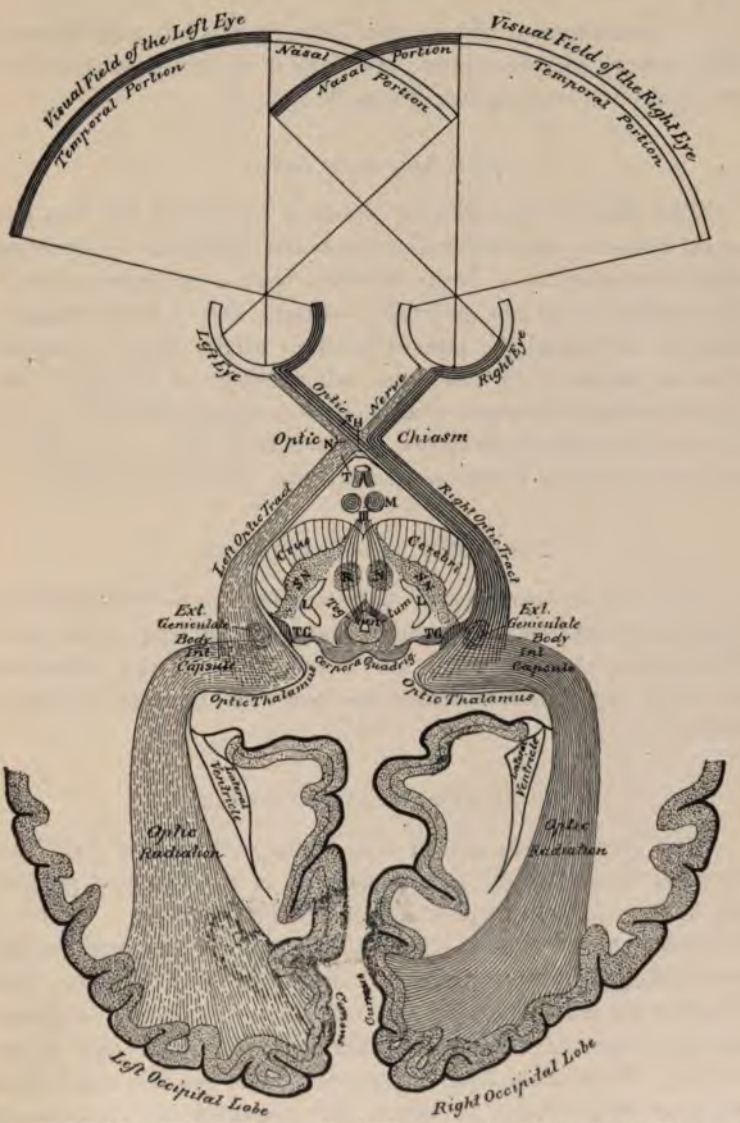


FIG. 14.—The Visual Tract. The result of a lesion anywhere between the chiasm and cuneus is to cause homonymous hemianopsia. III. Third nerve; S. N., substantia nigra; R. N., red nucleus of tegmentum; L., lemniscus; T. G., fibres from optic tract to corpora quadrigemina.

Irritation of the occipital lobe may cause hallucinations of light or even of objects which are referred to the opposite visual

field. Destruction of the occipital lobe on the left side destroys sight-memory pictures; hence recognition of things seen may be impossible, and reading power may be lost.

The Auditory Area.

The area of sensations of sound is located in the first and second temporal convolutions of the brain. Each ear is connected with both hemispheres; hence deafness from a unilateral lesion is only partial and is not generally noticed. But if both temporal lobes are destroyed the patient becomes totally deaf. Irritation of the temporal cortex may cause hallucinations of hearing. This may be the first symptom of a convulsion whose origin is an irritation in this part of the cortex. Destruction of the temporal cortex on the left side causes word-deafness. (See speech areas.)

The Smell and Taste Area.

The area of sensations of smell and taste is located at the tip of the temporal lobe on its under and inner surface, which rests on the sphenoid bone. Each lobe is related to sensory organs on both sides, and unilateral lesion does not often produce noticeable symptoms.

The Speech Areas and Aphasia.

The speech areas are of four kinds and in four locations. They are limited to the left hemisphere in right-handed persons and to the right hemisphere in left-handed persons. There is *the motor-speech area* in the posterior part of the third frontal convolution (Broca's convolution), in which the movements concerned in the act of speaking are controlled. The use of language and the power of talking are affected when this region is destroyed. There is the *auditory-speech area* in the first and second temporal convolutions, in which the memories of word-sounds are stored up. The understanding of language and the power of recollecting the names of objects are lost when this region is destroyed. There is *the visual-speech area* in the occipital and lower parietal region, in which the memories of printed words

are stored up. The understanding of written language and the power to read are lost when this region is destroyed. The *power of writing* is a part of speech and is usually lost when the motor-speech area is destroyed, but its exact location is not fully determined; some cases pointing to the second frontal convolution, others to the lower parietal convolution near the hand centre as its probable cortical position. (See Fig. 15.)

It is accepted that every word in ordinary use has a complex mental substratum, which may be termed the word-image, made up of a number of memory pictures. The memory of the sound of a word as spoken, the memory of the appearance of the word as printed, the memory of the muscular movements needed to write the word or to pronounce it, are known to be distinct from one another and yet to be associated together. Loss of one of these

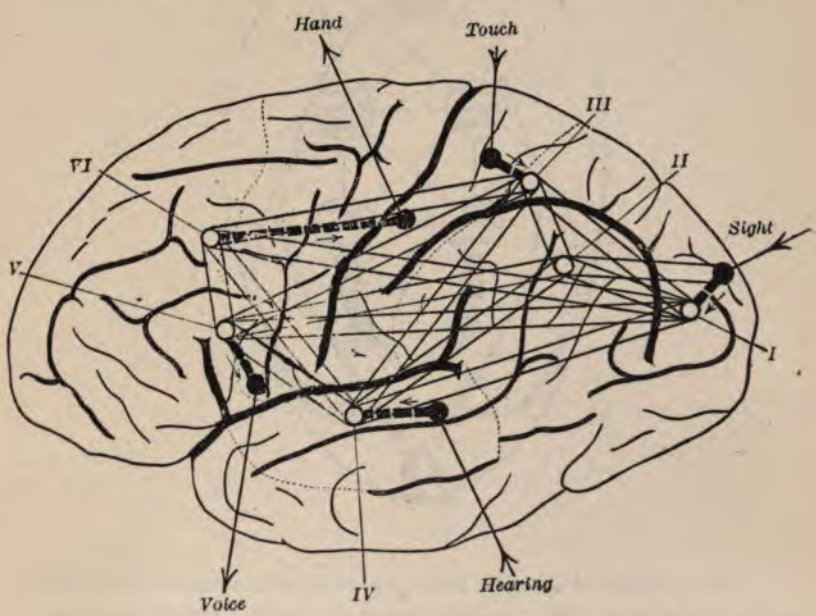


FIG. 15.—Diagram to illustrate Aphasia. The cortical sensory and motor centres are indicated by arrows. The secondary cortical centres of memories are indicated by circles. I. Visual of Objects. II. Visual of Words. III. Tactile. IV. Auditory. V. Speech. VI. Writing. These are joined to each other by association fibres which send impulses in both directions. Lesions of the association fibres as well as lesions of the cortex cause aphasia.

memory-pictures, or disturbance in their association, impairs the integrity of the word-image, and produces such defects in its use as are indicated by the names given to the respective varieties of aphasia. A division of disturbances of speech has been made into two great classes of sensory and motor aphasia—the first due to defect in the receptive, and the second due to impairment of the emissive functions of the brain. A further subdivision into several varieties has been made. These are word-deafness, word-blindness, agraphia, motor aphasia, and paraphasia. But, before considering these defects, let us look at the physical basis of the thought which lies back of the wor. Take the concept "bell" as an example.

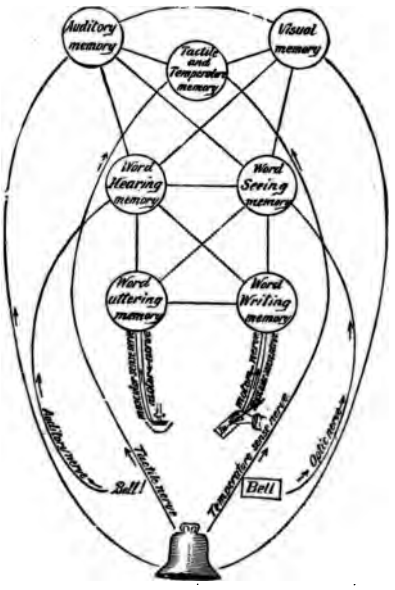


FIG. 16.

The concept of a bell is made up of separate memory-pictures of its shape, size, color (visual-picture); weight (muscular-sense picture); cold smooth surface (touch-picture); and sound (auditory-picture). These various memories, received by different senses and stored up in different parts of the cortex, are associated together in a mental image of the object bell.

To this mental image the word-image is added of the word bell as heard, as printed, as spoken, as written, making up a congeries of memory-pictures forming a "concept." Any one of these memory-pictures may be lost by disease. If the memories of the object are lost the symptom is called *apraxia.* If the memories of the word are lost the symptom is called *aphasia.*

To examine an aphasic thoroughly it is necessary to test—

1. The power to recognize objects seen, heard, felt, tasted, or smelt, and their use.

This will determine whether the condition of apraxia is present.

2. The power to recall the spoken name of objects seen, heard, handled, tasted, or smelt.

3. The power to understand speech and musical tunes.

4. The power to call to mind objects named.

This will test the integrity of the auditory-speeech area and of the association-tracts between other sensory areas and the temporal convolutions.

5. The power to understand printed or written words.

6. The power to read aloud and to understand what is read.

7. The power to recall objects whose names are seen.

8. The power to write spontaneously, and to write the names of objects seen, heard, etc.

9. The power to copy and to write at dictation.

10. The power to read understandingly what has been written.

These tests will determine the condition of the visual word-memories in the angular gyrus, and of the connections between this area and surrounding sensory and motor areas.

11. The power to speak voluntarily, and if it is lost, the character of its defects.

12. The power of repeating words after another.

This will test the integrity of Broca's centre and its association-tracts.

1. *Word-Deafness.*—If the memory of the sound of the word is lost, the word cannot be called to mind and cannot be recognized when heard. Show the patient a watch, and he is unable to name

it; tell him it is a stone, a match, a watch, and notice whether he dissents from the former and gives signs of satisfaction at the last. If he does, he has only auditory amnesia, but not word-deafness. If not, he is word-deaf, and is unable to understand what is said to him. The lesion lies in the posterior half of the first and second temporal convolution in the left hemisphere in right-handed, in the right, in left-handed persons. This is one variety of aphasia whose lesion is well known, though it rarely occurs alone.

2. *Word-Blindness.*—If the memory of the appearance of the word is lost, the visual image of it cannot be called to mind or recognized, and then the patient will be unable to write spontaneously, for he cannot remember how the letter looks which he wishes to write; and he will be unable to read, because the shapes of the letters and words seen arouse no recollection. As a matter of fact, words are forgotten more easily than letters, and if a patient is to relearn to read he must begin with letters and go on to words. Figures are sometimes recalled when words are forgotten, and many a patient can do mathematical calculations on paper who cannot read or write ordinary words. The reverse may also be true, the patient being able to read and write, but being unable to understand figures or calculate. Such patients may also play cards or other games, if they are not physically blind. It is not infrequently the case that persons who are thus word-blind can write at dictation, or copy, and yet show no evidence of understanding what has just been read or written. A distinction must be made between those who can and cannot do these things, though its pathological basis is still obscure. The condition of visual amnesia with word-blindness is due to a lesion involving the inferior parietal convolutions and angular gyrus, and is often associated with psychical blindness, but may occur independently of it. Word-deafness and word-blindness frequently occur together, and then the lesion is found involving both the temporal and the angular convolutions. Some cases of this condition have depended on a deep lesion in the temporo-occipital region, breaking the association-tracts.

3. *Motor Aphasia.*—If the memory of the effort needed to

pronounce a word is lost, a true paralysis of active speech occurs, though the muscles may not be weakened. This is the ordinary form of motor aphasia, due to a lesion of Broca's centre. It is to be noted that such a loss of speech involves a loss of the power of repeating words after another, as well as of voluntary speech, and is not accompanied by any inability to understand spoken or written language. In the uneducated, as in children, the acts of talking and writing are closely joined, as may be seen by watching the lips, which move in the act of writing. But among those accustomed to write much these acts are independent, and it is probable that many educated aphasics may be able to answer questions in writing when their efforts at speech fail. Reading aloud will also be lost in motor aphasia, for here, too, the inability to articulate hampers the patient.

4. *Agraphia.*—The independence of the effort-memory necessary for writing, and connected with movements of the hand from the effort-memories of speech, has been alluded to. When these are lost alone the condition is known as agraphia. In such a state the pen cannot be used. Copying, writing at dictation, and voluntary writing are all lost. It has been noted already that when a word cannot be called to mind, it of course cannot be written. But words can often then be written at dictation, if the person is one who writes much. Hence sensory agraphia and motor agraphia must be distinguished; the former being a part of word-blindness, the latter not at all associated with inability to read. The lesion of motor agraphia is unsettled, though a few facts point to the posterior part of the second frontal convolution as the probable seat of this function. It is not unlikely, however, that the more exact localization of fine movements of the thumb and fingers in the posterior central convolution by Horsley may be followed by the discovery of the writing centre in this vicinity.

5. *Paraphasia.*—These forms of aphasia are due to a loss of distinct memory-pictures. The several memory-pictures which are united in the word-image may thus be reasonably regarded as separate from one another in their location in the brain. But since they *are* joined together to form the word-image, it follows that the association-fibres joining the various areas are as necessary to the use of even a single word as the various areas with

FIG. 17.—The Association Fibres in the Centrum Ovale. A, Between adjacent convolutions; B, between frontal and occipital lobes; C, between frontal and temporal lobes, the cingulum; D, between temporal and frontal lobes—lesion of this tract causes paraphasia; E, between occipital and temporal lobes—lesion of this tract causes word-blindness; C.N., caudate nucleus; O.T., optic thalamus.

their memories. It is really by association only that an object or a word becomes a subject of thought or use. If these associations are broken, the result is a defect of language characterized by the misplacement of words, and the patient talks jargon.

There are many forms of paraphasia as there are many association-tracts, and it is not yet possible to assign different cases to their lesions.

It is evident that many differently located lesions will produce disturbances of speech. When the entire cortex is diseased —as in paresis—aphasia also appears.

Defects in the appreciation and use of music are called amusia, and present the same varieties as aphasia.

THE FRONTAL REGION.

There appears to be a certain relation between the frontal lobes of the brain and the higher forms of intellectual activity, the powers of fixing the attention and of reasoning and of self-control. But disease here does not cause a loss of any one mental faculty, and for the higher powers of the mind a general integrity

of the entire brain, not of any one part, is necessary. When it is considered that every concept is made up of numerous memory-pictures joined together, each of which has a separate location in the brain cortex, it becomes evident that to the process of thought a healthy state of the entire cortex is necessary, and also of the white matter beneath it, through which the associating fibres pass. And it is therefore impossible for a single lesion anywhere to cause a loss of memory or of imagination or of judgment. Yet for the co-ordination of facts into orderly series, for comparison, and for analysis of knowledge gained through the senses, the healthy state of the fontal lobes appears to be necessary. And lesions in the frontal region, especially upon the left side, are quite uniformly attended by mental dulness, apathy, lack of power of concentration, and imperfect self-control.

There are large areas of the cortex of the brain whose function is undetermined. These are much more extensive on the right hemisphere than on the left. There are no definite symptoms produced, so far as we now know, by lesions in these areas; but the negative fact is certain, that lesions in them do not cause disturbances of motion, of sensation, or of speech.

The cortex of the hemispheres upon the base of the brain lying on the orbital plate, on the sphenoid and temporal bones, and on the tentorium cerebelli, has as yet no assignable functions, and lesions in these regions do not produce recognizable symptoms.

Symptoms of Cortical Disease.

In the study of cases of cortical disease, it is necessary at the outset to distinguish general from local symptoms, for it is the latter only which enable one to locate the lesion. *General Symptoms* are those which are common to various kinds of brain disease in whatever region they are located. They are headache, vertigo, digestive disturbances, general convulsions, optic neuritis, with or without blindness, delirium, and coma. These are due either to an increase of the intracranial contents (as by the growth of a tumor) and consequent presssure, or to some other interference with the normal condition in the brain. They give no evi-

dence as to the exact region of the brain which is affected. They merely indicate that the brain is diseased.

Local Symptoms, on the other hand, depend entirely for their production upon the region of the cortex which is invaded. They are disturbances of motion and of sensation of various kinds, and of sensory perception; disturbances of memory; and loss of speech. They may be divided into symptoms of irritation and symptoms of destruction; and it is not infrequently the case that the former precede the latter in the course of a disease. An irritative lesion produces symptoms due to an increased activity of the area affected; *e. g.*, spasms, pain, tingling and numbness, flashes of light, sounds, or hallucinations of smell and taste. A destructive lesion produces symptoms due to a loss of function in the area involved; *e. g.*, paralysis, anæsthesia, blindness, deafness, loss of smell and taste, and of the use of language. Each of these points to a different location of the disease, and is essential to its localization.

II. Tracts Within the Brain.

The Association-Tracts.

The first system of fibres in the centrum ovale is the *association* system. (See Fig. 17.)

It can be shown by careful dissection that each convolution is joined to the two adjacent convolutions by fibres which pass around the separating fissures. Also, that bundles of fibres exist which pass from each convolution to the convolution next but one, and so on. Hence, it may be stated that each convolution has a possible connection with every other. Besides this association of convolutions by small bundles of fibres, it is possible to find a distinct set of association-tracts which pass between more or less distant regions. One such tract passes from the frontal lobe, collecting its bundles from all three convolutions, backward to the occipital lobe. Another tract joins the occipital with the anterior part of the temporal lobe. Another passes from the upper two temporal convolutions forward to the third frontal convolution, passing beneath the island of Reil. And a tract from the frontal to the posterior temporal area may also be found.

The function of these association-fibres is to form the physical basis for the association of concrete memories and of psychical acts, and their integrity is necessary to thought. By studying subjectively the association of ideas in the mind the importance of their function is evident. When they are destroyed by disease various forms of paraphasia appear.

The Commissural Tracts.

The second system of fibres in the centrum ovale is the *commissural* system. This joins corresponding areas of the two hemispheres with one another. The commissural fibres between the frontal, parietal, and occipital lobes of the two sides pass in the corpus callosum. Those from the temporal lobes pass in the anterior commissure. The function of these fibres is to harmonize the action of the two hemispheres. Simultaneous movements of like nature can be made with great facility with both upper extremities. Movements which are difficult when attempted with the left hand alone become easy when associated with corresponding movements of the right hand—as, for example, drawing a circle, writing one's name. It is the commissural fibres joining the motor convolutions only which can be thus tested. Failure to perform easily corresponding bilateral motions in face, hands, or feet would indicate some obstruction to conduction in them.

The Projection-Fibres.

The *projection* system includes those fibres which join a definite area of the cortex with parts of the nervous system lying below it, viz., some nervous mechanism in the basal ganglia, brain axis, or spinal cord. Indirectly, through the medium of such mechanisms, the external world is projected upon the brain and reaches consciousness, and voluntary impulses originating in the brain are sent to the muscles. The fibres gather together within the hemisphere at the upper level of the basal ganglia, and either end in the optic thalamus or go on between these ganglia through the internal capsule to the brain axis and spinal cord.

The majority of these fibres end in the optic thalamus, which

FIG. 18.—The Projection Tracts Joining the Cortex with Lower Nerve Centres. Sagittal section showing the arrangement of tracts in the internal capsule. A, Tract from the frontal lobe to the pons, thence to the cerebellar hemisphere of the opposite side; B, motor tract from the central convolutions to the facial nucleus in the pons and to the spinal cord; its decussation is indicated at K; C, sensory tract from posterior columns of the cord, through the posterior part of the medulla, pons, crus, and capsule to the parietal lobe; D, visual tract from the optic thalamus (O.T.) to the occipital lobe; E, auditory tract from the internal geniculate body (to which a tract passes from the VIII. nerve nucleus [J] to the temporal lobe; F, superior cerebellar peduncle; G, middle cerebellar peduncle; H, inferior cerebellar peduncle; CN, caudate nucleus; C.Q., corpora quadrigemina; Vt., fourth ventricle. The numerals refer to the cranial nerves.

is thus connected with all parts of the cortex of the brain. Of the function of these we know very little. Two large bundles, however, are separable from the mass.

The Visual Tract.—One of these passes inward and forward from the occipital lobe, and joins the pulvinar of the thalamus and the external geniculate body. This is *the visual tract.* (Fig. 14.)

The optic nerves decussate partially at the chiasm, so that

each optic tract contains fibres from both retinæ. Each tract passes to the optic thalamus, external geniculate body, and anterior corpus quadrigeminum. Thence the fibres of the visual tract pass out, to turn backward through the internal capsule and through the centrum ovale to the cortex of the occipital lobe. A lesion anywhere in this course causes homonymous hemianopsia.

The Auditory Tract.—A second bundle passes from the temporal lobe upward to the thalamus, internal geniculate body, and posterior corpus quadrigeminum, in which the auditory tract from the acoustic nucleus ends. This conveys impulses of sound from both ears to each temporal lobe, and is the *auditory tract.* These are the only bundles of the thalamic radiations whose function is determined. (Fig. 18 E.)

Some of the projection-fibres pass on through the internal capsule without communicating with the basal ganglia, and of these we know three distinct bundles. (Fig. 18 A.)

The first is collected from the three convolutions of the frontal lobe, and passing between the caudate and lenticular nuclei in the anterior division of the interal capsule, descends in it to the base of the brain, and issuing in the inner third of the foot of the crus cerebri, passes down to the pons, where it terminates in nuclei lying in the ventral half. The nuclei thus reached by these fibres are also joined by other fibres from both hemispheres of the cerebellum, which enter the pons at its lateral surfaces in the middle peduncles. Thus it is evident that a connection exists between each frontal lobe and both cerebellar hemispheres, the crossed connection being greater than the direct one. Of the function of this tract we know nothing.

The Motor Tract.—The second bundle of the projection system is *the motor tract.* It comes from the posterior part of the third frontal convolution, the two central convolutions, and from the paracentral lobule, and passes out of the base through the middle third of the crus cerebri. Its fibres collect at the middle portion of the upper surface of the internal capsule, those from the lower parts of the cortex passing straight inward, those from the upper parts curving outward and downward to pass around the side of the lateral ventricle. Thus within the centrum ovale,

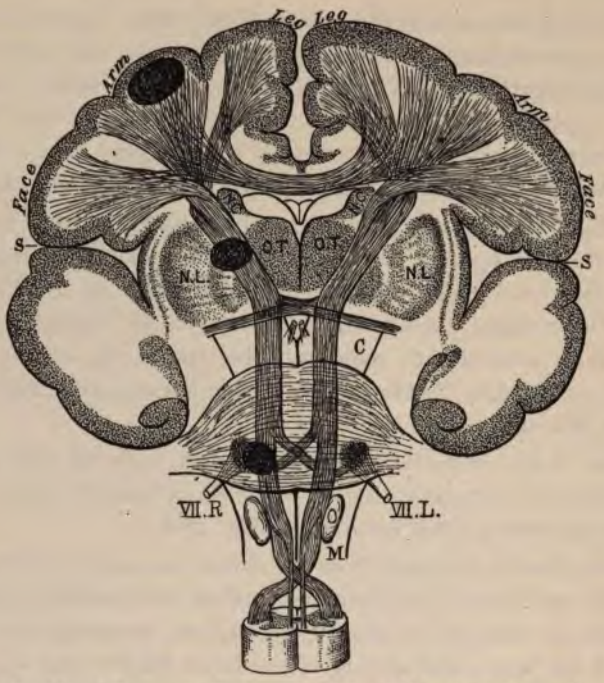


FIG. 19.—The Motor Tract. S, Fissure of Sylvius; N.L., lenticular nucleus; O.T., optic thalamus; N.C., caudate nucleus; C, crus; P, pons; M, medulla; O, olivary body. The tracts for face, arm, and leg gather in the capsule and pass together to the lower pons, where the face fibres cross to the opposite VII. nerve nucleus, while the others pass on to the lower medulla where they partially decussate, to enter the lateral columns of the cord; the non-decussating fibres pass to the anterior median columns. The effect of a lesion situated at three points in the tract is shown on the left side of the figure.

these fibres, if looked at from in front, appear like the sticks of a fan, and like those sticks their relative position is altered in the point of junction, where those passing inward from the lowest part of the cortex lie in front of those which pass downward from its upper part. Thus in the capsule the order from before backward is, *first,* the fibres conveying speech impulses to the pons and medulla; *second,* the fibres conveying facial-motor impulses to the pons; *third,* the fibres destined to the arm centres of the cord; *fourth,* the fibres transmitting impulses to the leg centres in the cord. The fibres conveying impulses to the muscles of the trunk lie between those to the arm and leg. (See Fig. 20.)

From the anterior half of the posterior division of the capsule this tract passes through the middle third of each crus, through the pons (where the division to the facial nucleus crosses to the opposite side and ends), and thence by way of the pyramids of the medulla to the crossed pyramidal and direct anterior median columns of the spinal cord. It is evident, however, that the concentration of this tract is much greater in the capsule than in the centrum ovale, where the individual fibres are scattered among the other systems and occupy but a small area from before backward.

Lesions beneath the third frontal convolution of the left side produce motor aphasia. Lesions beneath the central convolutions in the centrum ovale produce paralysis, which will vary according to the position of the lesion. The nearer the lesion lies to the cortex the more will the symptoms resemble those of cortical disease, monoplegia being the rule. The nearer the lesion to the point of junction at the capsule the more will the symptoms resemble those of capsule lesion, hemiplegia being the rule. When the motor tract is injured by a lesion in the lower half of the pons, the result is paralysis of the face on the side of the lesion and of the arm and leg on the opposite side (alternating paralysis).

The Sensory Tracts.—The third set of fibres of the projection system include those which lie just posterior to the motor tract, and which pass inward from the parietal convolutions. These take a similar course to those of the motor tract, and fill up to a considerable extent the space between it and the radiation of the visual tract, toward the occipital lobe. They are mingled with fibres which pass to the optic thalamus, but are separable from them in fœtal brains, and may be traced down through the capsule to the tegmentum of the crus, where they divide into a portion going to the lemniscus, and a portion going to the formatio reticularis. The fibres can be traced through these tracts downward to the medulla and through the sensory decussation to the posterior pyramids of the medulla in which the posterior columns of the spinal cord begin. See Figs. 21-24.) This set of fibres conveys the sensations of touch, pain, temperature, and muscular sense, and lesions in its course will cause disturbance of these sensations.

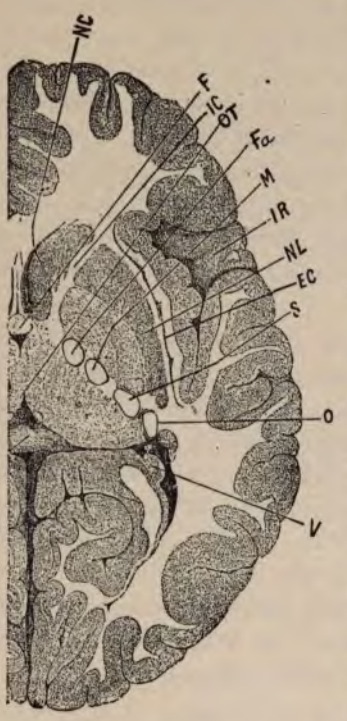


FIG. 20.—Horizontal Section through the Brain. (After Strumpell.) *NC*, nucleus caudatus; *F*, fornix; *IC*, anterior half of internal capsule; *OT*, optic thalamus; *Fa*, facial tract; *M*, motor tract; *S*, sensory tract; *O*, visual tract, in posterior half of internal capsule; *IR*, island of Reil; *NL* nucleus lenticularis; *EC*, external capsule; *V*, lateral ventricle, posterior cornu.

Like lesions in the motor tract, the rule obtains that the nearer the cortex the more likely is the lesion to cause an affection of a single limb, while the nearer the capsule the more likely is the symptom produced to be hemianæsthesia. A lesion in the lower half of the pons may produce alternating hemianæsthesia, the face being anæsthetic on the side of the lesion, and the limbs on the opposite side.

From these facts it is evident that a lesion which lies in the centrum ovale, at any point posterior to the præcentral fissure of the frontal lobe, may produce recognizable symptoms, for it must affect either the motor, or the sensory, or the visual, or the auditory tracts, or individual fibres of those tracts.

III. The Basal Ganglia.

The symptoms produced by lesions in the basal ganglia—corpora striata and optic thalamus—are not to be distinguised from those caused by lesions of the projection-tracts which lie near them in the internal capsule.

Lesions of the corpora quadrigemina cause nystagmus, blindness, and ophthalmoplegia.

IV. The Cerebellum.

The cerebellum is the central organ of equilibrium. It is joined to the spinal cord by the direct cerebellar column, which

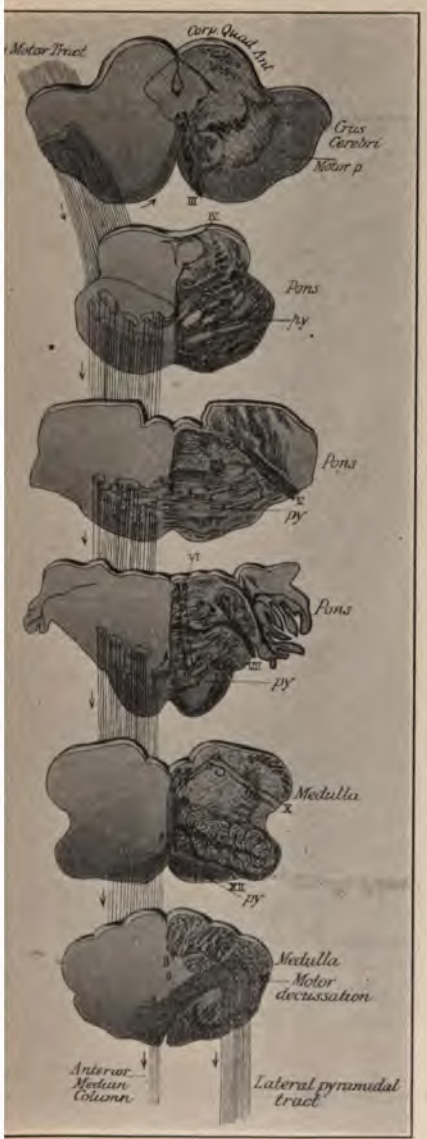


FIG. 21

...e Motor Tract in the Cerebral Axis.

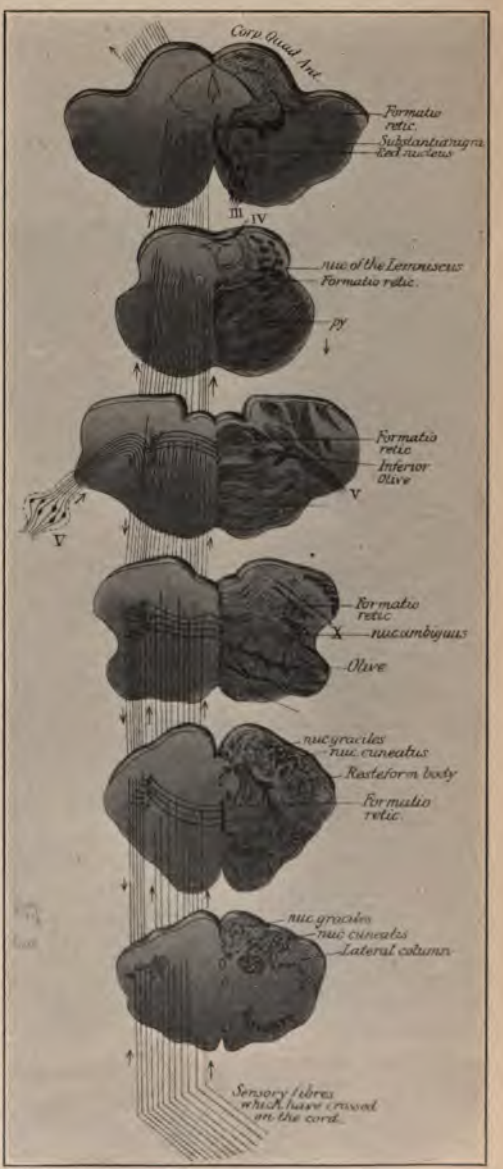


FIG. 22

The Tract of the Tactile Sense.

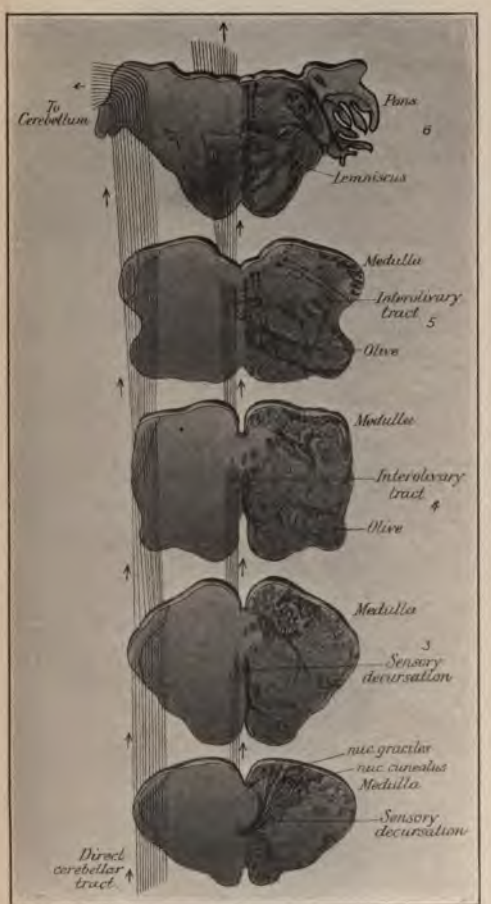


FIG. 23

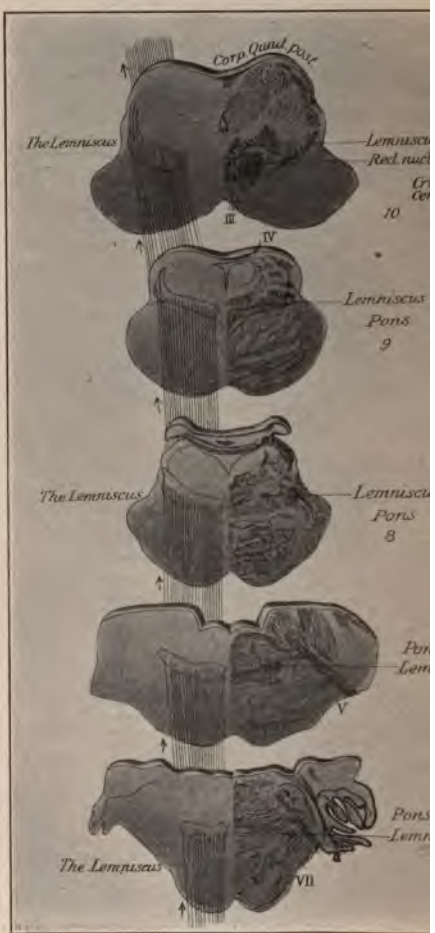


FIG. 24

The Tract of Muscular Sense.

carries sensory impulses up to it through the inferior peduncle. It is joined to the motor tract by the middle peduncle, which conveys impulses to the pons and thence to the spinal cord, directing balancing motions. It is joined to the cerebral hemispheres both by the middle and superior peduncles, whose exact functions are unknown.

Lesions of the cerebellum, or of its peduncles, cause staggering gait. This differs from that in locomotor ataxia, for there is no ataxia of the limbs when the patient lies down, giving support to the body. When the middle peduncle is irritated the patient staggers toward the lesion; when it is destroyed he staggers away from the lesion. Lesions of the cerebellum also produce vertigo.

V. The Cerebral Axis.

The various long tracts which join the brain to the spinal cord pass through the crus cerebri, pons, and medulla near to the nuclei of origin of the cranial nerves. These tracts are shown in Figs. 21-24. The cranial nerve nuclei were shown in Fig. 7.

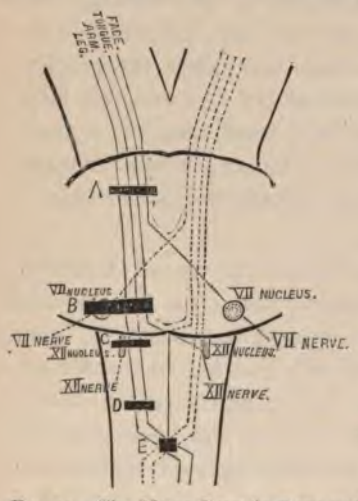


Fig. 25.—The Motor Tract in Pons and Medulla. Lesion at *A* causes hemiplegia of side opposite to lesion; lesion at *B* causes alternating paralysis; lesion at *C* causes paralysis of tongue on side of lesion and of extremities of opposite side; lesion at *D* causes paralysis of extremities of opposite sides; lesion at *E* causes paralysis of extremities of both sides.

Lesions of the cerebral axis, such as small hemorrhages, small areas of softening, or tumors destroy these tracts and also these nuclei. The tracts as a rule decussate on entering the cord, hence a unilateral lesion of the cerebral axis causes motor or sensory symptoms in the extremities of the opposite side The nuclei of the cranial nerves send those nerves directly outward without decussating; hence, a unilateral lesion of the cerebral axis causes motor or sensory symptoms in the face on the same side. From the combination of these two sets of symptoms it is easy to diagnosticate a lesion of the cerebral axis. (See Fig. 25.)

THE CEREBRAL DISEASES OF THE VASCULAR ORIGIN.

HEMORRHAGE, EMBOLISM, THROMBOSIS, "APOPLEXY."

The most common cerebral diseases are those due to a rupture of a vessel in the brain, *cerebral hemorrhage,* or to a stoppage in a cerebral vessel, *cerebral embolism* and *cerebral thrombosis.*

The cerebral circulation. See "Quain's Anatomy."

Pathology.—The condition which leads to such diseases is chronic endarteritis; a chronic inflammatory process in the intima and muscular coats of the vessels with a production of new cellular tissue which may (1) go on until it obliterates the lumen of the vessel, producing thrombosis; or (2) undergo calcareous degeneration, roughening the wall and inducing fibrin deposits, which may occlude the vessel, producing thrombosis, or be washed onward, producing embolism; or (3) undergo fatty degeneration, thus eroding the intima and weakening the muscular coat, causing aneurisms (miliary or larger) which rupture, producing cerebral hemorrhage. See Delafield and Prudden's "Pathology."

Pathology of Cerebral Hemorrhage.—The vessel ruptures. The blood spreads out in the membranes, filling the fissures and depressing the cortex, or if the hemorrhage is subcortical, tears the surrounding brain. A clot forms, thin and flat on the cortex, globular in the brain, which tears and compresses the brain-tissue around it. This clot gradually shrinks and undergoes absorption, leaving a hæmatin stain; the disintegrated brain-tissue undergoes fatty degeneration and absorption. There remains a mass of softened tissue without or with a connective-tissue wall, or finally a cyst, or a mass of sclerotic tissue or merely a scar. The torn brain never reunites. From the point of laceration degeneration begins, which extends along the various tracts, *e. g.,*

descending degeneration in motor tract through the cerebral axis and spinal cord, or ascending degeneration in sensory tracts to the cortex. When the pressure is removed the cortex may resume its function or the uninjured tracts may resume their functions. Hence partial recovery is the rule after cerebral hemorrhage.

Pathology of Cerebral Embolism or Thrombosis.—The vessel being occluded, the brain-tissue which is supplied with blood through it is cut off from its nutrition. The first arrest of blood supply is extensive, but when collateral circulation is established the final area or region which softens may be small. In the cortex the collateral supply by anastomosing vessels is extensive. In the basal ganglia and capsule it is very imperfect. Hence the permanent effect of occlusion is more serious in the arteries entering the base than in the small branches in the cortex. If large vessels in the cortex, *e. g.*, a main branch of a Sylvian artery, is plugged, the area of softening may be extensive. A fatty degeneration and a necrosis of brain-tissue follow the occlusion of the vessel. In the softened tissue there are at first extravasations of venous blood (red softening); later these are absorbed and the tissue in a stage of fatty degeneration is yellow (yellow softening). If there is simple necrosis without extravasation or fatty degeneration, the brain has its natural color but is soft (white softening). The softened tissue contracts, leaving a depression, or a cavity, or a scar; in some cases a cyst with smooth walls and serous contents.

The Situation of the Lesion.—Cerebral hemorrhage: in basal ganglia involving the internal capsule in fifty per cent., next in frequency in centrum ovale, cortex, pons, and ventricles; very rarely in cerebellum.

Cerebral embolism, forty-nine per cent. left; forty-one per cent. right; ten per cent. both hemispheres. Central ganglia and capsule, sixty per cent.; next in frequency cortex supplied by middle cerebral artery, (1) motor area, (2) aphasic area, (3) visual area. Least frequent pons and anterior or posterior cerebral arteries.

Cerebral thrombosis is middle cerebral, basilar, vertebral, and anterior and posterior cerebral vessels in order of frequency named.

Etiology.—Thrombosis is common below age of forty-five;

hemorrhage after forty-five; age of maximum liability to hemorrhage, fifty to sixty. Males more liable than females to hemorrhage. Predisposing causes of endarteritis are gout, lithæmia, alcoholism, and syphilis; associated diseases are nephritis, endocarditis, and emphysema. Anything which increases the heart action may cause cerebral hemorrhage, e. g., fright, anger, exertion, cold bath. Anything which weakens its action may cause thrombosis.

Symptoms.—Preliminary: For months before the onset, vertigo, insomnia, headache, inattention, and imperfect memory. Temporary attacks of numbness in one-half of the body.

Sudden onset, "apoplexy," loss of consciousness. The result depends on the size and rapidity of hemorrhage, or on the size of the vessel occluded.

Paralysis of one-half of the body (hemiplegia), with coma and convulsions, is the ordinary form. Face, red; pulse, slow, high tension, full; respiration, deep, stertorous. Temperature normal, but after some hours high, 102° to 104° F. Deviation of eyes and head may occur in large lesions; the patient looks toward the side of lesion. Pupils vary. Inequality is most important symptom, usually dilated on side of lesion. Hemiplegia, hemianæsthesia, and hemianopsia, at first. Reflexes suspended; inhibited by irritation for a short time, then exaggerated permanently. Rigidity of limbs when hemorrhage is large or into the ventricle; convulsion when hemorrhage is on cortex or in ventricle. Urine retained; contains albumen and sugar, or may be passed involuntarily.

Death may occur in one or two days, in coma, with temperature of 108° F.

Usually there is a slow recovery of consciousness and permanent paralysis, which gradually decreases during a year, and then remains stationary.

In lighter cases there may be no loss of consciousness, but partial hemiplegia or arrest of any cortical function.

The permanent symptoms a week after the acute onset indicate the situation of the lesion. The symptoms which remain at the end of a month indicate its extent; *e. g.*, hemiplegia with hemi-

anæsthesia and hemianopsia at first; and permanent paralysis, not total, in arm and leg only, without sensory affection finally. In some cases there is a development after two months of contractures in the paralyzed limbs. This indicates the existence of descending degeneration in the motor tract.

Course.—After the acute onset, in which the patient is confined to bed for two weeks to two months, depending on the severity, the improvement begins; the chief symptom being hemiplegia. The face is flat on the paralyzed side, and is slightly drawn by the healthy muscles away from the paralyzed side so that the mouth is crooked. The eye can be closed (*vs.* facial palsy), and can be moved (*vs.* ophthalmoplegia). The tongue deviates toward the paralyzed side. The finer motions of the hand and arm are more permanently affected than elbow or shoulder motions; and the upper extremity is carried in a flexed position. The leg is stiff and extended, does not bend easily at the knee, and the foot is dragged on its inner edge and swung around in walking. The muscles of the trunk and respiration are very rarely affected. The bladder and rectum may not be under full control. There may be a diminution of sensation in the paralyzed side, and possibly hemianopsia. Hemianæsthesia or hemianopsia may remain without hemiplegia. A total loss of all senses, smell, taste, sight, hearing, and touch on one side only, occurs in hysteria only. The tendon reflexes are usually much increased on the paralyzed side, and are somewhat increased on the other side as well. The contractures which develop may cause pain, because the limbs are rigid. Otherwise there is no pain.

Aphasia of some kind often accompanies right hemiplegia, or may occur alone. (See page 56.)

Some mental symptoms are usually observed after an apoplexy, and may be the only permanent symptoms. There may be a lack of control of the emotions, so that the patient laughs or cries with little cause; an irritability of temper; a lack of judgment; imperfect memory of recent events; imperfect power of concentrated attention; general bewilderment, so that the patient does not recognize his surroundings or friends; mild delirium or delusions; or even dementia. These usually pass away gradually, but sometimes remain.

The various symptoms, motor, sensory, aphasic, or mental, depend on the situation of the lesion, and enable a conclusion to be arrived at regarding it.

In any case epileptiform attacks may develop, and continue at long intervals after an apoplexy.

Diagnosis. Cerebral Hemorrhage.—In old persons with atheromatous arteries and hypertrophied heart. Sudden onset of coma during exertion or in excitement. Coma deepens; temperature falls to 97° to 95° F. in an hour; after three hours rises to 101° F. Gradual recovery of consciousness in three to five days, with permanent symptoms of hemiplegia.

Cerebral Embolism.—At any age with heart disease, or after childbirth. Sudden onset with no loss of consciousness or slight condition of mental confusion, or rapid return of consciousness. Temperature does not fall, but may rise to 102° F. Improvement occurs within twenty-four hours to a marked degree, but symptoms return in three or four days and are permanent, either monoplegia, or hemiplegia, or aphasia. Jacksonian epilepsy may develop after a few months.

Cerebral Thrombosis.—At any age, but chiefly in syphilitic persons and in middle-aged men. Premonitions usually occur. Slower onset without coma, but dulness of mind. Consciousness returns soon, if lost. Temperature does not fall; may rise to 100° F. The paralysis resembles that in embolism.

The diagnosis between these three conditions is never positive.

Causes of coma other than apoplexy: Syncope, epilepsy, uræmia, diabetes, alcoholism, opium poisoning, sunstroke, cerebral injury. Apopletic coma is deep and patient cannot be aroused. Face is congested, eyes turned to one side, pupils unequal, paralysis is unilateral, limbs may be rigid; appearance indicates previous convulsions. Physical examination reveals absence of injury and presence of predisposing disease, *e. g.*, atheroma, endarteritis, cardiac disease, albuminuria, syphilitic scars. The absence of diagnostic signs of other causes of coma confirms the diagnosis of apoplexy. Reflex movements can be caused on non-paralyzed side by producing pain. The tendon reflexes are often

lost at the onset. The cremasteric reflex is always lost on the paralyzed side.

Prognosis depends on the depth of coma, on the condition of pulse and respiration, on the constitution and age of the patient, and on the number of previous attacks.

Treatment in stage of apoplexy: Rest; quiet; ice-bag to head; venesection in plethoric cases; purgatives of a drastic nature; stimulants if heart failure is present.

For hemiplegia: Exercise; massage; faradism to the muscles for purpose of exercise.

Medicines are of no use in either stage.

Cerebral Atrophy.

Cases of cerebral disease in children, either unilateral or bilateral, present some peculiar features of three different types.

First, cases of hemiplegia with or without athetosis.

Secondly, cases of mental defects of various grades.

Thirdly, cases of sensory defect of different types. Epileptiform seizures of *petit mal* or of *grand mal* type occur frequently in patients who may be assigned to any one of these groups.

I. *Infantile Hemiplegia.*—The *symptoms* are the sudden development of a unilateral paralysis after a series of convulsions attended by high fever and its attendant discomforts, and a period of unconsciousness of varying duration; then a gradual improvement in the paralysis after the active manifestations of the onset have subsided; and finally, a stationary condition, in which the face is but slightly affected in its voluntary or automatic movements; the speech is usually regained, if it had been lost; the arm is quite seriously paralyzed, the fingers being stiff and awkward, and sometimes being in constant slow involuntary motion (athetosis); the leg is held rather rigid, so that the child limps in walking, and often develops a club-foot. There are, of course, on the one hand, light cases in which the symptoms finally amount merely to clumsiness in the fingers. There are, on the other hand, severe cases in which a double hemiplegia has occurred, both halves of the body being equally affected, and in which both arms are useless and both legs so stiff, so closely adducted, and so

helpless, that walking is impossible (diplegia). The latter are cases in which all the symptoms date from birth. In all cases the paralyzed limbs are found to be affected in their growth and development, so that they are smaller, colder, stiffer, and weaker than the others; the reflexes are exaggerated, but the electrical reactions are not qualitatively changed, and the sensation is normal. This condition remains through life as a permanent defect, and although the division of contractured muscles or tendons and the application of ingenious apparatus may correct deformities and make the paralyzed parts fairly useful, and although the application of electricity to the muscles chiefly affected may increase their nutrition and this prevent contracture which comes from the unbalanced strain between the various muscles, yet any great degree of improvement is impossible. In more than one-half of these cases epileptic attacks are of frequent occurrence.

The cases which date from birth must be divided into those in which there has been evidence of traumatism during labor, and those in which there was none. In the former class it may be stated confidently that cerebral hemorrhage, usually meningeal, is the cause of the symptoms. In the latter class it is probable that an intra-uterine encephalitis or some unknown cause has prevented the fœtal brain from developing.

The cases which have developed suddenly after birth are cases in which the ordinary causes of hemiplegia in adults have been acting, and must be traced either to encephalitis, hemorrhage, or embolism, or disease of the vessels. The degree of spontaneous recovery in any case can be pretty well determined by an examination at the end of the second year. A complete cure never occurs.

The fits in organic epilepsy are likely to be more frequent and severe than in idiopathic epilepsy; as many as twenty convulsions may occur daily for weeks at a time. These fits do not destroy life, but render life a burden. When, however, they are infrequent they may be benefited to some degree by the use of bromides.

II. The second class of cases presents mental defects more noticeable than physical symptoms. The child may be slow in

learning to talk, may seem unable to fix its attention upon anything continuously, may be exceedingly active, in constant motion—the activity being, however, aimless; may throw things about, or tear things up, or put everything into its mouth; may be very difficult to manage because of its inability to retain and combine impressions with sufficient power to reason upon them; and may, therefore, be incapable of appreciating the meaning of punishment, if this be inflicted. Such children may have good powers of perception, may recognize persons and objects, show pleasure at bright colors, or music, or caresses, but fail to show evidence of thought in the sense of reasoning power, judgment, or self-control. Some patients constantly drule at the mouth, cannot be taught cleanly habits, and are manifestly imbecile. Other patients are quite bright in many directions, may even be precocious, show talents in music, or drawing, or fondness for mathematics, designing, languages; yet are apparently unable to appreciate moral ideas, cannot be taught to tell the truth, are cruel and bad, will not control any of their impulses, and so are the distress and despair of parents and teachers. It is those mental qualities which are the product of the highest evolution which have failed to develop in this class of cases. The final result is that they have to be taken care of all their lives, either at home or by attendants, being incapable of supporting themselves or directing their conduct. Many of them have epilepsy. Some of them have hemiplegia.

III. The third class of cases is less common than the two preceding, and is likely to escape observation unless carefully investigated. The patients belonging to this class may be hemiplegic or may present no motor or mental defects, though they may be the subjects of epilepsy. They do have defects of sensory perception. Such defect may be in the form of hemianopsia or of deafness. It is probable that many cases of deafmutism belong to this class, and in some persons, as in Laura Bridgeman, deafness, dumbness, and blindness coincide.

The pathological changes in the brain in these various cases differ widely, and are as follows, in the order of frequency:

Porencephalus, a localized atrophy or agenesis, leaving a

cavity in the cerebral hemisphere, which may be deep enough to open into the ventricle.

Sclerotic atrophy, an atrophic condition of the brain with an increase of connective tissue and disappearance of the nervous elements; affecting both hemispheres, or one only, or a part of one only; or limited to small areas in various parts.

Maldevelopment and apparent atrophic condition of the minute structures of the hemispheres, chiefly cortical, the cells resembling those of a new-born child, but with no apparent gross defects in the brain.

Atrophy, consequent upon the condition of softening produced by embolism or thrombosis, and limited in extent to certain arterial districts of the brain.

Meningo-encephalitis, a condition shown by thickening and adhesion between the pia and the brain, with destruction of the cerebral cells and atrophy of the cortex.

Cysts lying on the brain and producing atrophy by pressure or associated with atrophy due to the original lesion of which the cyst remains as a trace.

Hemorrhage on or in the brain, as shown by the remains of a clot, or by hæmatin staining of a cyst, of the pia, or of the sclerotic tissue.

Hydrocephalus with extreme dilation of the ventricles, so that the brain tissue is reduced to a mere wall about the cavity.

These are the conditions found at death in cases presenting the clinical features just studied. It is evident that they have this in common, namely, a condition of atrophy of the brain. The origin of this atrophy is not always clear. In some cases it is clearly congenital and due to a maldevelopment of the embryo.

In other cases it is clearly traceable to injuries at birth. In other cases it must be ascribed to affections of various kinds, such as inflammations of the membranes or of the brain substance, or vascular lesions and their consequences, such as occur in adults. But it is evident that the various processes of disease have as a fairly uniform result a condition of atrophy with sclerosis of the brain, which may be termed sclerotic atrophy. The

differenec in the clinical types is due to the varying situation of the lesion rather than to its varying nature.

For such conditions there can be no treatment.

Tumors of the Brain.

Varieties in order of frequency:

Gumma: soft gelatinous, or hard cellular, situated on base or in cortex, usually beginning in meninges.

Tubercular: hard masses, often multiple, especially frequent in children, situated anywhere, sometimes primary, usually secondary. This is the variety in more than one-half the cases in children.

Glioma: peculiar to the nervous system, cells and fibres like the neuroglia, usually infiltrated and without boundaries, but single, usually vascular; hence symptoms vary and hemorrhage occurs; usually situated in the white substance.

Sarcoma of all varieties: fibro-, round-cell, spindle-cell, glio-sarcoma, begin from connective tissue of membranes as a rule, sometimes in the brain. Hard single tumors, of various sizes, easily separable and well defined, situated on base, in meninges, in cerebellum.

Carcinoma: rare, usually secondary.

Occasional varieties are fibromata, osteomata, psammomata, cholesteatomata, angiomata, lipomata, ecchinococcus, and cysticercus.

Aneurisms of the base give rise to symptoms of tumor.

For Pathology of Tumors, see Delafield and Prudden's "Pathology."

Causes of Brain Tumor.—1. Predisposing: Sex: males twice as frequently affected as females. Age: children most liable, as tubercular tumors are frequent. Young adults next. Old age is quite exempt, except from carcinoma.

2. Exciting. Injuries. Severe emotional shock. Unknown in large majority.

Situation.—Cerebral cortex, twenty-five per cent.; cerebellum, twenty-five per cent.; centrum ovale, fifteen per cent.; basal

ganglia, ten per cent.; pons, ten per cent.; crus and corpora quadrigemina, ten per cent. Others, five per cent.

Effects of Brain Tumor.—1. Direct effects. Irritation of gray matter with nervous discharges, and destruction of brain-tissue.

2. Indirect effects. General pressure on entire brain. This may be limited by the falx or tentorium. It produces hydrocephalus by pressure on ventricle or veins of Galen. Vascular disturbance by pressure on arteries and veins. Meningitis, localized and not purulent, except in tubercular cases. Thinning of cranial bones.

Effects of increased pressure are seen after death in flattening of the convolutions, anæmia and pallor of the brain, dry sticky state of membranes, and distension of ventricles by serum.

Symptoms of Brain Tumor.—I. General symptoms depend on existence of a new growth independent of its position.

Headache in ninety-five per cent., constant or intermittent; general or local; dull and deep, increased by anything which deranges the cerebral circulation. Sometimes head is tender to percussion at certain points; may have evidence of pain even in state of stupor; it may be agonizing; it may give feeling of band about head; it is often worse at night, causing insomnia, especially in gumma.

Mental disturbance, eighty-five per cent. Dulness and apathy, with imperfect attention and memory. Depression and emotional state. Slow speech, gradual mental failure, childishness, and dementia; fainting attacks, stupor, and coma in the last stage. Occasionally delirium and active insanity.

Optic neuritis, eighty per cent. Venous congestion. Swelling of papilla, loss of disc outline; radiating appearance about the disc and hemorrhage; then optic-nerve atrophy leaving white or gray disc. In cases of subcortical tumor of small size it may be absent. Sight may be affected; visual field small and irregular, or unimpaired for a time.

Vertigo or sense of giddiness, frequent in tumors in posterior fossa. May occur in tumors anywhere on change of position. May be due to paralysis of one or more ocular muscles.

Vomiting, with or without nausea or relation to food. Frequent in tumors in posterior fossa.

General convulsions of epileptic type. *Petit mal* or *grand mal.*

Nystagmus, lateral oscillation of eyes. Frequent in disease of corpora quadrigemina and cerebellum.

Loss of control over sphincters occurs, especially with tumors in the frontal lobes.

Glycosuria and polyuria, especially with tumors in the posterior fossa.

Slow pulse; 50 to 60.

II. Local symptoms dependent upon the position of the tumor.

Frontal Lobes: Mental dulness, lack of control of emotions, irritability, inattention, and childishness, dementia, loss of sense of smell.

Left third frontal convolution. Motor aphasia.

Central Convolutions, and motor tract. Monospasm and monoplegia when in or near cortex. Hemispasm and hemiplegia when deep or in capsule. Sometimes slight anæthesia in paralyzed part.

Occipital Lobe and visual tract. Hemianopsia, noticed by patient as unilateral blindness. Visual hallucinations and flashes of light on one side.

Left Temporal first and second convolutions. Sensory aphasia: form, word-deafness.

Left Lower Parietal lobule and angular gyrus or in occipito-temporal tract. Sensory aphasia: form, word-blindness.

Base of brain.

Anterior fossa: Loss of smell.

Middle fossa: Hemianopsia, paralysis of ocular muscles, III., IV., VI.; neuralgia of face and anæthesia, V.; paralysis of III. and opposite hemiplegia, neuroparalytic ophthalmia, nystagmus; bilateral spastic paralysis, deafness.

Posterior fossa: Paralysis of VII., IX., X., XI., XII.; vertigo and aural symptoms; cerebellar ataxia, tendency to one side; paralysis widespread from medulla disease.

Cerebellum: Vertigo, vomiting, cerebellar ataxia when middle lobe is involved; headache, frontal or occipital and severe; optic neuritis early.

Diagnosis.—Cerebral symptoms: general alone, or general and local, point to the presence of a tumor.

Differentiation from chronic nephritis: By examination of urine; by condition of heart, arteries, and blood-pressure; by appearance of optic disc and retina in many cases, hemorrhages more frequent and white spots far from disc; by character of headache, more severe in tumor; by age of patient, young person = tumor.

From hypermetropia with astigmatism and anæmia, with menstrual disorder: By examination of eyes and relief by glasses; by cure of anæmia by diet, iron, and aloes; by slight degree of headache and neuritis.

From hysteria with migraine: By study of the patient and separation of real symptoms; by condition of the optic disc.

From abcess: By history of trauma or of otitis media; by severity of early symptoms in abscess; by fever in abscess; by late development of optic neuritis, rare in abscess.

To determine the location: Study carefully the local symptoms, "signal symptom," *e. g.*, numbness and twitching of thumb. Study also the order of appearance of local symptoms: thumb, hand, face. Compare the several local symptoms, paralysis + aphasia, or + hemianopsia.

To determine the variety: Estimate probable frequency of various tumors at different ages. Look for history of syphilis, tuberculosis, cancer, or sarcoma. Study rapidity of onset; glioma, tubercle, cancer are rapid. Study signs of irritation; more frequent in glioma and meningeal tumors. Study signs of variation in severity of symptoms; more variation in glioma. Study situation of the tumor; basal tumors are often gumma, tubercle sarcoma. Intracerebral tumors are often glioma, sarcoma. Peculiar forms of hemiopia and early optic neuritis indicate sarcoma of pituitary body or aneurism.

Course of the disease: Begins with general symptoms usually. Sometimes begins with local spasm or a general convulsion. Gradual increase in severity and number of symptoms.

Finally death in stupor or in general convulsion or suddenly, from heart failure, or recovery under specific treatment.

Duration is from one to three years.

Prognosis bad, excepting in gumma and when tumor can be removed.

Treatment.—For gumma: Inunctions of mercury and KI to three hundred grains daily.

Surgical treatment: Removal is possible when diagnosis of location is exact. There is danger of return.

For Cranio-Cerebral Topography, see page 49.

Abscess in the Brain.

Acute encephalitis with formation of pus results in an abscess. The abscess may extend rapidly, not being encapsulated, and cause death; or it may become encapsulated and develop slowly, or become stationary and remain for years as a foreign substance producing no symptoms, but finally enlarge or break. Occasionally multiple abscesses develop in the brain secondary to pyæmia, producing cerebral symptoms in the course of that disease. There may be a secondary meningitis in either form.

Situation.—Is usually in the temporo-sphenoid lobe or in the frontal lobe or in the cerebellum, in the white matter.

Causes.—Chronic otitis media, forty per cent. Fracture of cranial bones, thirty-five per cent. Chronic rhinitis, ten per cent. Caries of any cranial bone, ten per cent. Secondary to infectious diseases, five per cent.

Symptoms.—After an injury or after otorrhœa or other cause cerebral symptoms develop suddenly; severe headache, vomiting, mental distress and dulness, stupor, a chill, a high temperature for a day or two; leucocytosis, possibly convulsions; rarely local symptoms; hemiplegia, hemianopsia, aphasia, cerebellar staggering. In a week the patient is very ill, with irregular temperature, sometimes with chills, sometimes no fever; slow pulse; intense pain, delirium, stupor, and optic neuritis. If abscess extends death follows in two weeks, or suddenly from rupture. If abscess discharges into ear or nose, or if it can be opened, symptoms subside. Rarely symptoms subside spontaneously slowly,

the abscess becoming encapsulated and latent, and patient apparently recovers, but is subject to headache and vertigo; and has a relapse later.

Diagnosis.—

Abscess	*vs.*	Meningitis.
Temperature normal or not high.		Temperature high without remission.
Pulse slow.		Pulse rapid.
Patient in stupor.		Patient irritable, all senses over-acute.
Spasms and rigidity rare.		Spasms and rigidity frequent.
Cranial nerves not involved.		Cranial nerves involved early.
Optic neuritis frequent.		Optic neuritis rare.

Abscess	*vs.*	Thrombosis of Lateral Sinus.
Temperature normal or not high.		Temperature high with remissions.
Pulse slow.		Pulse rapid, weak.
Chill at onset not repeated.		Chills frequently repeated.
Little sweating.		Profuse sweating.
Head tender to percussion?		Pain over mastoid and jugular.
		Thrombi in lungs.

Diagnosis of location is made partly from the exciting cause and partly from the local symptoms.

Prognosis fatal, unless abscess can be opened.

Treatment.—In the few cases in which the abscess can be located, operation of trephining and drainage.

Cerebro-spinal Sclerosis.

Multiple or disseminated sclerosis is a rare disease affecting the brain and spinal cord together. Small regions of sclerotic tissue are found irregularly scattered through the entire nervous system, without destruction of the axis cylinders of the nerve-tracts. Its cause is unknown.

Symptoms.—Headache, vertigo, malaise, mental irritability, inattention, imperfect memory, lack of self-control, and inability to work. Later, tremor of hands increased by an effort to hold them still, nystagmus, increased knee-jerk, stiff spastic gait, weakness, and finally general tremor on effort. Slow speech, each

syllable separately enunciated without variation of tone. Optic atrophy. Dementia and attacks of epilepsy and hemiplegia may finally occur.

Course is exceedingly irregular, symptoms may disappear for months and then return. The disease is often mistaken for hysteria.

Diagnosis is made by excluding all other organic diseases, by nystagmus, tremor, and speech.

Treatment.—Arsenic $\frac{1}{50}$ gr. daily; solanin $\frac{1}{2}$ gr. daily, increased.

Syphilis of the Brain.

Syphilitic affections of the nervous system may be produced by:

1st. Syphilitic endarteritis.

2d. Direct action of the syphilitic poison upon the nervous system.

3d. Syphilitic exudations in the meninges.

4th. Syphilitic deposits in the brain itself.

5th. Hereditary syphilis.

1st. Syphilitic endarteritis produces a progressive diminution in the calibre of the blood-vessels, resulting in a state of anæmia, malnutrition of the brain supplied by the vessels affected, and leads to thrombosis followed by localized softening in the area cut off from its blood supply. The symptoms of this condition are in the early stage those of malnutrition of the brain and imperfect action in the regions affected. The patients present at first symptoms of neurasthenia, and later slight temporary suspension of functions, such as temporary aphasia, numbness in one limb or in one-half of the body, a condition of weakness not amounting to paralysis in one limb or one-half of the body, double vision, or vertigo. Stimulants to the heart relieve these symptoms, but after a time if a thrombosis occurs the symptoms of cerebral apoplexy suddenly appear. After such an apoplectic attack a partial recovery may ensue, but as a spot of softening in the brain is usually left, a complete recovery is not to be expected; hence the fact that a syphilitic lesion which can be re-

moved by treatment is the cause of the apoplectic attack does not warrant a more favorable prognosis in such cases.

2d. The toxic effects of syphilis on the nervous system are manifested by general disturbances of function, which produce the symptoms of neurasthenia of all the various types. As a rule, in syphilitic neurasthenia the symptoms appear to be worse toward evening and insomnia is more persistent than any other symptom; hence in general neurasthenia with insomnia any syphilitic element must be looked for and treated before the neurasthenia can be cured. Neurasthenia may occur in syphilitic persons without any direct connection with the syphilis, which at the time may be latent. A test of treatment is the only one enabling differential diagnosis to be made.

3d. Syphilitic exudations into the meninges of the brain and syphilitic meningitis are exceedingly common as a sequel of syphilitic disease. From two to ten years after the infection gummy exudations may occur in any part of the brain, but are more frequent upon the base of the brain and about the crus and pons. The glue-like substance is deposited rapidly and extensively through the meninges, producing pressure upon the subjacent brain or upon the cranial nerves, and thus causing suspension of function in the parts compressed. Occasionally the syphilitic exudation takes the form of a hard tumor, which then produces the regular symptoms of tumor of the brain. The symptoms of syphilitic gumma or syphilitic basilar meningitis are headache, worse at night, insomnia, mental irritability, vertigo, distressing sensations in the head, occasional vomiting, and local symptoms of paralysis of the cranial nerves, the third and sixth being the ones particularly affected. If the exudation is not upon the base, or occurs in the convexity or over the cerebellum, the local symptoms will be those of irritation or of arrest of function of the particular portion of the brain affected. Thus spasms and paralysis, paræsthesia and anæthesia, hemiopia, or any variety of aphasia may be caused by the disease. The pressure upon the vessels produced by syphilitic exudations adds to the complexity of the symptoms. Convulsions are frequently observed in such cases, both of a general and of a local type. Increased thirst and hunger and polyuria are often found. The course of the case

is more rapid in its onset than that of brain tumor, but resembles it in all other respects and may be associated with optic neuritis. The symptoms subside rapidly under inunctions of mercury and large doses of iodide of potash, and hence prognosis is good in the majority of cases, though relapses are frequent.

4th. Syphilitic deposits in the brain itself may be of the nature of small dessimated spots, producing chronic indurative or sclerotic processes of small regions of softening. The symptoms of this affection are identical with those of general paresis, and can only be distinguished from paresis by the result of treatment.

5th. Hereditary syphilis occasionally produces the symptoms of cerebral atrophy or the symptoms of multiple cerebro-spinal sclerosis in children, and in either of these conditions this fact must be kept in mind, and if other signs of hereditary syphilis are discovered this element must be considered in the treatment.

FUNCTIONAL NERVOUS DISEASES.

NEURASTHENIA.

Nervous prostration, nerve tire, nervous irritable weakness is a functional disease of the entire nervous system, brain, spinal cord, spinal and sympathetic nerves, dependent upon malnutrition, characterized by nervous weakness and irritability.

A balance between expenditure of energy and nutrition must be preserved.

Brain overwork or anxiety;

Spinal cord overwork by long marches or sexual overaction.

Nerve overwork by various occupations in which a single act is repeated;

Vasomotor work in maintaining vascular tone and nutritive processes, all require a sound basis and rest at periods.

When balance between store and expenditure of energy is broken, exhaustion results. Then nature enforces rest by fatigue, by sleep. One may disregard nature, but if so one must pay the penalty.

Causes.—I. Excessive expenditure of nerve energy.

Primary neurasthenia more common than secondary.

Influences favoring its production:

1. Bad hereditary influences. Weak nervous system.
2. Feebleness in childhood, with poor nervous system.
3. Wrong methods of training and education.
4. The struggle for existence. Kant's rule, eight hours for work, eight hours for diversion, eight hours for sleep.
5. Anxiety, worry, mental depression, fear.
6. Mental or physical overwork.
7. Sexual excesses.

II. Deficient supply of nervous energy.

Secondary neurasthenia.

Influences favoring its production:

1. Weakening diseases of all kinds of organic nature.
2. Indigestion and dyspepsia, with auto-infection by products.
3. Gout, rheumatism, uric-acid diathesis.
4. Infectious diseases, typhoid, grippe, malaria.
5. Alcoholism or abuse of drugs.

Result of the action of these causes is to produce malnutrition of the nervous system and state of irritable weakness.

1. The power of continuous activity is impaired; weakness results; hence, mental work is imperfect; attention flags; memory fails; ambition ceases; physical activity is imperfect; exercise tires one; lassitude; vegetative functions are impaired. Digestion is poor; constipation occurs; vascular tone is impaired; heart feeble. Balance must be maintained between blood supply and functional activity. When this is impaired work is impossible. Secretions are changed in amount and character, *e. g.,* urine.

2. The power of mental and physical control is impaired and irritability results; inhibition may be partly automatic and partly conscious; it must be conscious and voluntary to prevent morbid fear and apprehension; influence of fear is to inhibit all nervous acts—*e. g.,* walking on ice, oral examination, stage fright; this control is weakened, and hence irritability results.

Symptoms of Neurasthenia.—

1. Cerebral: Headache—dull, occipital, frontal; sensitiveness of scalp; vertigo; insomnia; cerebral sensations—fulness, band, pulsation.

2. Mental: Apprehension—morbid fears, of places, people, solitude, etc.; inattention and forgetfulness; incapacity for work; idiosyncracies become prominent; irritability of temper ensues; anxiety and exhaustion; imperfect respiration and rapid pulse.

3. Spinal: Pain, especially in nape of neck and sacrum; irritation; hypersensitiveness; sensitiveness about ribs and intercostal nerves; weakness, legs give out; sexual irritation, erections, impaired power; emissions; bladder irritability, frequent micturition.

4. Vasomotor: Flushes, cold extremities; undue sweating;

transient blueness or œdema; *tache cérébrale* of Trousseau; tachycardia; palpitation.

5. Gastro-intestinal: Indigestion and dyspepsia, especially acid stomach; imperfect liver action; constipation; pseudomembranous colitis; distension of abdomen with gas.

6. Sensory and motor symptoms: Indefinite pains and paræsthesia; joint affections; apparent paralysis—no change in electric activity; vision imperfect; asthenopia; visual field may be retracted. Other senses rarely affected; tinnitus.

Diagnosis made by the existence of a cause; the absence of objective signs of organic disease; the temperament of the patient; the variability of symptoms; the disproportion between complaint and actual power.

Treatment.—Absolute necessity of establishing nerve-energy by restoring balance between store and expenditure. Hence supply nutritive elements to nervous system by proper food, properly digested; by increasing circulatory power; by exercise; by hydrotherapy; by massage; and diminish expenditure of nervous energy by rest, if necessary absolute, and by diversion.

Direct treatment may be by tonics of all kinds; by sedatives, if patient is extremely nervous; by electricity; galvanism. Treat symptoms only when absolutely necessary. Combat the desire of these patients for drugs, and their dependence on stimulants and opiates.

Hysteria.

A functional disturbance of the nervous system characterized by mental and moral perversion, lack of self-conrtol, and disorders of any or all the bodily functions.

Women, between fifteen and twenty-five years, or at any age.

Men, at any age, and even children, may be affected.

Causes.—Hereditary influence very marked; malnutrition of the nervous system from anæmia; indigestion; from bad habits of life; emotional excitement; fear, anxiety, jealousy; tendency to imitation yielded to; epidemics of hysteria; uterine and ovarian irritation is a rare cause. Traumatism, especially accidents attended by great fear. Several causes may concur.

Symptoms.—1. Mental changes, and moral perversion.

Increased irritability, and emotional excitability with impaired self-control.

Excessive emotion leads to exhaustion.

Craving for notice and sympathy.

All sorts of means taken. Self-injury; sores which won't heal; needles swallowed; blood sucked and vomited; animals concealed.

Purposeless acts of criminal kind, like reasoning mania.

Hallucinations may occur.

Depression is not uncommon, and melancholia may be feared. But in melancholia facial expression is characteristic, thought is slow, delusions are of self-accusation, weight is rapidly lost.

2. Disorders of various functions:

(1) *Motor.*—Spasms, contractures, paralysis, convulsions, globus hystericus, vomiting, diarrhœa, vesical irritation, convulsion attended by sobbing and crying, lasts longer than an epileptic fit, does not begin or end suddenly, and tongue is not bitten, and there is no asphyxia. Paralysis, varies in intensity, does not affect the face in hemiplegia, no typical gait, no bedsore, no incontinence; aphonia, aphasia, cough.

(2) *Sensory.*—Hyperæsthesia of special senses, dislike of light or noises; hyperæsthesia of skin, spinal irritation; hyperæsthesia of limbs, numbness; hyperæsthesia of ribs, pressure of dress; hyperæsthesia of ovaries; hyperæsthesia of bladder, frequent micturition; pain anywhere, rarely definitely localized; especially in joints, hip, knee, ankle, wrist, elbow; fixation, atrophy of muscles; joints easily moved under ether; no deformity; anæsthesia to pain, usually localized, in skin or mucous membranes; unilateral loss of special senses.

(3) *Vasomotor.*—Palpitation and faints; variations of arterial tone, flashes, œdema; hæmatemesis, hæmoptysis; small hemorrhagic spots, stigmata; rises of temperature, 105° to 110° F.

(4) *Secretion.*—Saliva; gastric juice varies. Urine; large or small amount.

Hysterical attacks: Crying and laughing, produced by emo-

tional strain; scream, convulsion, opisthotonus, facial expression, series of short attacks, fit of crying, passes water, cataleptic rigidity, hysteroepilepsy.

The character of the symptoms varies greatly. The symptoms appear and disappear suddenly.

The course of the disease: Chronic condition with periods of intensity; cure, sudden and marvellous; great variation in symptoms; very rarely death in convulsion or from suicide.

Diagnosis is never made from a single symptom, but from a study of the general condition.

Treatment.—Mental change and moral education; hydrotherapy; the rest cure; mental therapeutics; tonics, strychnine, iron, and quinine.

Symptomatic remedies:

Sedatives for the nervous state, bromides, valerian, lavender, asafœtida. Opium is to be avoided, for habit is easily formed.

For convulsion, prevent injury, don't use chloroform, use cold water, hold patient, stop respiration, don't press ovaries, give emetic.

For faints, irritation over heart by ice, internally spirits ammonia, or lavender.

For spasms, hot or cold applications, cautery.

For paralysis, electricity, especially faradism.

For pain, hypodermics of water after one of morphine, local applications of sedatives.

Epilepsy.

Epilepsy is a functional neurosis characterized by sudden transient loss of consciousness with spasmodic actions of the muscles.

There are three varieties.

(1) *Petit mal,* in which the loss of consciousness is very brief and the spasm slight or absent.

(2) *Grand mal,* in which there is a coma, a general convulsion, with biting of the tongue, frothing at the mouth, some-

times involuntary micturition, and a general weakness and a deep sleep following the coma.

(3) Psychical attacks in which a sudden mental aberration occurs with acts of violence, or odd actions lasting several hours or even days. In all the varieties the patient is unconscious during the attack, no matter what he does, has afterward no memory of what occurred in the attack, and is often unaware that he has had any attack at all.

Etiology.—It is more common in males than in females. It is a disease of early life, but no age is exempt. Many cases can be traced to some injury or organic disease of the brain in infancy or childhood. Any organic brain disease may cause epilepsy in adult life. It is a frequent equal of injuries of the head or brain. It is also of toxic origin; chronic alcoholism, lead poisoning, tobacco poisoning, syphilis, or any of the infectious diseases may be followed by epilepsy. In nervous children intestinal toxaemia may cause it. Peripheral irritation long continued in a very nervous person may cause it; *e. g.*, foreign bodies in the nose or ear, irritation of teething, adenoids in the throat, phimosis, painful scars. In the majority of cases no cause can be found. In the aged arterial sclerosis and multiple areas of cerebral softening are a cause.

Theory of the Disease.—The nerve cells store up energy which is liberated in any act of mind or will in a moderate degree and is accompanied by sensations or muscular acts. In epilepsy it is supposed that this energy is suddenly liberated without moderation and hence sensations are forced upon consciousness (the aura) and muscles are thrown into violent action (the convulsion). All expenditure of nerve energy is followed by exhaustion until the supply is renewed. Hence the coma, sleep and weakness following the attack. The reason why nerve cells in some persons are ready to discharge their energy spontaneously or on slight provocation is unknown. Any mental or physical strain is capable of setting off the explosion. Many poisons in the blood are also capable of doing so. The reason bromide and other sedatives check the disease is because they reduce the tendency to the sudden discharge.

Symptoms.—The attack is often preceded by some sensation, a numbness, gastric sensation, a vertigo, a flash of light, a sound, a fixed idea. This is called an aura. It is an indication of the organ of the body or of its corresponding region of the brain in which the irritation begins. The convulsion may be general or localized in one part, of the latter it indicates some irritation in the motor area of the brain. Thus many tumors begin with a local spasm or a general fit. The attack is followed by a sleep which is evidence of exhaustion of the brain. Every possible variation in the severity of attack from a petit mal of an instant's duration to a grand mal of many hours has been observed. When a series of grand mal attacks occur the condition is termed status epilepticus. This is the only dangerous form, as death rarely occurs in an attack. The pupils of epileptics are always dilated and though they contract again dilate in light. The memory becomes poor as the disease goes on. Judgment is impaired. A final state of dementia is observed in many cases.

Prognosis.—Recoveries are rare. The disease is chronic, but the intervals between attacks may be lengthened by treatment.

Treatment.—A simple diet from which meat is almost excluded. Sufficient amount of bromide of potassium and sodium and ammonium combined to hold attacks in check, given on rising and at bed time well diluted. Other remedies are tincture of simulo, one teaspoonful; antipyrin, 15 grains; solanum carolinense, 20 drops; chloral, 10 grains; Belladonna, Digitalis. Heart tonics may be combined with all these remedies. Bromide acne is to be treated by arsenic and hot baths. When bromide causes delirium, as it may, it is to be avoided.

Chorea.

Chorea, or St. Vitus Dance, is a functional neurosis characterized by sudden quick twitching of the muscles.

Etiology.—It follows rheumatism in 30 per cent. of the cases. It is preceded or attended by endocarditis and valvular disease in 25 per cent. of the cases. It follows mental shocks

in 40 per cent. of the cases. It is often attended by anæmia. It occurs in the spring time after the house confinement of winter. It is more common among the poorly nourished. It is a disease of childhood, but adults occasionally are affected.

Symptoms.—The twitching may be unilateral or bilateral. It may affect the face chiefly, or this may escape. It leads to grimaces and difficulty of talking. It may be so intense as to necessitate care in bed. Usually it is of slight degree. It may cause apparent ataxia and weakness in the muscles affected, but is not followed by paralysis. Heart murmurs are found in many cases, sometimes anæmic and transient, often organic. Anæmia is often present. The child is often very irritable and fretful. The course of the case is slow, from three to six months. Relapses are very frequent.

Prognosis is good for recovery, but bad for recurrence.

Treatment.—Arsenic gradually increased to point of tolerance; antipyrin or exalgin in moderate dose according to age.